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# COOPERATION IN THE CARE OF THE PATIENT*

BY ELLIOTT P. JOSLIN, M.D.†

FOREWORD. My predecessors have presented so adequately, so convincingly and so appealingly the privileges and duties of all physicians to the physical and humanitarian needs of the individual patient that common sense directs me to tread a somewhat different path and to point out how the care of the patient includes the care of his family and active coöperation of all their physicians; and I shall also attempt to describe what I like to think of as The Hospital Family and its place in our medical midst. I was strengthened in this decision by the words of President Conant in his keynote address at the 150th Anniversary of the founding of the Harvard Medical School when he interpreted present trends in medicine and indicated that discoveries in medicine in the future were far more likely to originate in groups of workers than to come from a single investigation. So, too, I believe that the time has arrived when the care of the patient is linked with the care of his entire family and, to secure satisfactory results all his medical attendants must unite in the task and this they can do most advantageously in association with a hospital.

*The Diabetic, his Family and his Doctor.* While exploring with you my thesis that the care of the patient includes that of his family and close contacts among his doctors, I shall draw freely upon diabetes as an illustrative disease. I am sure you will agree with me that it is appropriate to the occasion. And this is not simply because I think diabetes a good disease and because I think the diabetic somewhat of a superior being and that his outstanding physical and mental precocity should be cultivated, or for the reason that I have more experience with diabetes than I do with other diseases, but first, because diabetes is preëminently a disease of the latter half of life, that half upon which our medical efforts have made the least impression, secondly, because it is an hereditary disease and thus is lodged in and transmitted by the family in contrast to all save one of the major contagious diseases, thirdly, because it is a coming disease with so growing a frequency that the 1930 prophecy of Dr. Louis I. Dublin that it would equal tuberculosis in importance in the space of 10 years is already more than approaching fulfillment, and fourthly, because diabetes is the disease *par excellence* which demands that doctor, surgeon, obstetrician, pediatrician, all the specialists and all our skillful laboratory friends work together as a unit both with the patient and his family to treat it. Fortunate it is that the diabetic more than repays us for all the pains we take with him, because his very body has served as an enticing and productive laboratory for the elucidation of many problems of intermediary metabolism and presents an opportunity, better than any other of which I know, for the investigation of the premature advent of old age.

*The Diabetic's Family.* Naunyn knew that diabetes was hereditary. He demonstrated heredity in 18 per cent of his patients. His whole theory of diabetes was based upon the hereditary background of the disease and he explained its early quiescence and its later appearance in adult life as a result of the play of accessory factors upon this inborn tendency. What a wise and thoughtful man was this Naunyn—the Nestor of Diabetes. Today's diabetic child proves that Naunyn's contention was true. Originally the diabetic child's heredity appeared to be a scant 20 per cent and was often reported to be less, but that was due to his former brevity of life and to poor history taking. Naunyn really had the vision when he recognized the principle that in dealing with heredity it is "seek and ye shall find, knock and it shall be opened unto you". Today longevity replaces brevity of duration in the child and now at the end of only ten years since the discovery of insulin, his first chance for a decade of a diabetic life, Priscilla White has proved his heredity to be 53 per cent, a figure also reached by Grote for 65 of his diabetic children. But there were some who rightly said that all the tenets of genetics were not fulfilled and therefore we went across the river to Cambridge to secure the advice of Professor Crozier of the Department of General Physiology in Harvard University and through him the direct assistance of Professor Gregory Pincus and this resulted in the genetic proof that diabetes is hereditary in the Mendelian recessive sense. Not content with the original 500 diabetic families studied, Pincus and White confirmed their

*Lecture delivered at the Harvard Medical School, November 16, 1933, on The Care of the Patient.

†Joslin, Elliott P.—Medical Director, George F. Baker Clinic of the New England Deaconess Hospital. For record and address of author see "This Week's Issue," page 656.

conclusions, first, by the addition of some 300 families (I stopped the series here remembering Professor Zinsser's warning of the futility of sitting on a boiled egg) and secondly, more fruitfully by the direct examination of 27 families of conjugal diabetics and various other family groups both diabetic and non-diabetic as well, the latter to serve as controls. In this task I cannot avoid the satisfaction of having interested the specialized knowledge of Harvard University in a clinical problem.

What are the applications of all this diabetic polyverbiage and diabetic heredity to the Care of the Patient? As a matter of fact the acceptance of the principle of diabetic heredity has changed our tactics in our warfare to defeat diabetes. At present we concentrate our efforts on the 400,000 patients in the United States with the disease, and the far smaller number of families which they represent, rather than dissipate our energies upon 125,000,000 citizens who do not have it and 98 per cent of whom we know never will have it. With more complacence and pity we can view the fat lady and fat man on the street, because, perhaps, they have no diabetic heredity. On the other hand our zeal to educate the family of the diabetic is redoubled. In season and out of season we preach to the diabetic family the danger of two diabetics marrying one another and raising a 100 per cent brood of diabetics. It is only fair to say that we temper this pronouncement with the reassuring statement that if they do marry one another and do have 100 children, the last three of the hundred will not exhibit their diabetes until past the age of seventy, and the safer suggestion that if they would only be content to put up with non-diabetics for partners, at least their immediate children should be free from the disease. Not content with data thus far acquired upon the inheritance of diabetes, this last summer, Dr. White with the courage of her convictions spent her Sunday afternoons hunting in diabetic families and actually found among 105 relatives of our patients eight wholly new and undiscovered cases. And what does this signify?

The unknown patient, the unknown diabetic, thus became recognized and so could receive in the incipiency of his disease proper care. I think we do not half realize that the cases of a disease we don't see and therefore do not treat are the important cases. The carriers of typhoid bacilli, streptococci, tubercle bacilli and spirochaetae are the most dangerous enemies there are to those about them and, undetected, their respective diseases hurry these often innocent carriers to their death. These are the symptomless diabetic patients, often brought to light by insurance companies, and for whom I have incontrovertible evidence from my own records that their prognosis is better than that of the diabetic with symptoms, 42 per cent better in the younger decades of life, 38 per cent better in the middle decades, and even in the oldest decades 31 per cent better.

How fascinating the hunt for the unknown diabetic in order to give him the attention he deserves! How can we recognize him? Must we depend upon sheer hard work alone, repeated and systematic examinations of the urines and bloods of all the members of all diabetic families? I trust not for long. Only the other day President Lowell warned against slavery to routine and contrasted it with imagination, ". . . imagination. Ah, that is the essential, how to stimulate the imagination." We concede that diabetes is hereditary. Granted, but is there not some trait, some quality in the newborn child which may give a hint as to whether he is liable, if he lives to his full three score years and ten, to develop the disease? Perhaps there is and in fact there may be several stigmata. Priscilla White found that diabetic children at the onset of their disease were 2.2 inches taller than the ordinary child; and because diabetic children are often precocious we are waiting for the physiologists and endocrinologists and all of you to describe a method by which an excess of the pituitary element can be recognized in any human being. Already we perceive the importance of the pituitary factor in the diabetic problem, because on the one hand diabetes is frequent in pituitary disease and on the other develops when the pituitary is active, at adolescence, during pregnancy, at the menopause, and the frequency at this latter period very likely accounts for the doubling of the incidence of diabetes in women as compared with men above the age of 45 years. Who has sought for a diabetogenic element at birth? When Professor Pincus asked so simple a question as to the frequency of the various blood groupings in diabetic families I could only plead ignorance. Do you know? We need some one across the Charles to ask us questions. What is the diabetogenic factor which is transmitted with the gene? How will you reveal it? If we only knew which children in a family were prone to diabetes we would be doubly solicitous for them.

Some of the predisposing causes to the outbreak of diabetes in a family can be foreseen and averted. Mr. Meade of the Lincoln National Life Insurance Company long ago directed attention to the fact that if one eliminated the influence of obesity, the incidence of diabetes remained the same throughout life. It is quite unnecessary for the members in a diabetic's family to be or to become fat. My statistics show that fifty per cent of the women who develop the disease at the age of 45 years or above are 30 per cent overweight. The last 1226 diabetic female patients above the age of 45 years weighed 181 pounds. Certainly these are pretty fat ladies. Priesel and Wagner would dub them the blamable diabetics as opposed to the blameless type of diabetes in the child. But can we dismiss the subject so easily? Will not some-

one couple the more reproachful words blamable doctors to the words blamable diabetics? For some time the doctor has not been excused if he simply treated his patient for smallpox, diphtheria and typhoid, but did not protect the family. I feel that the same responsibility exists for making an attempt to prevent diabetes in a diabetic's family or at least to make the attempt to diagnose it early in its course. We seek to uncover the beginnings of cancer and are trying hard to report tuberculosis, years and months rather than days and hours before the patient dies, and we know that we shall never be successful unless we realize that the care of the patient includes the care of the family. To a far less degree than the plainly evident finger-pointing signs toward diabetes of heredity and obesity are the cautionary signals of hyperthyroidism, biliary disease and infections which to some extent favor the development of the diabetic predisposition. On the other hand liberal muscular development is antagonistic to it.

*The Public Health Nurse.* I rather think the public health nurses have outdistanced us doctors in including the family in the care of the patient. A generation ago I confess there were so many patients to see in the "District" that scant attention was paid by me to the families. Originally the nurse simply nursed the patient, but times have changed and I suspect that a good share of the time of the modern nurse is taken up with the family of the patient. Sometimes certain of the medical profession think this has been overdone, but it may be that if these same doctors had a nurse in their home at the arrival of a new baby, they would not consider it amiss if the nurse found time to give a bath or two to the two and three year olds or confined to his room the first born just returning from school with a sore throat or sent still another with a pain in his stomach to bed, supperless, until the medical parent returned. Who would say that a nurse did her duty while caring for a patient, no matter what the type of his disease, if she did not report to the family doctor a possible case of tuberculosis in a brother or sister, wife or parent? Would she be excusable if she did not report suspected gonorrhea, syphilis or scarlet fever in the family of a woman approaching confinement? If a Public Health Nurse, must she not observe the circumstances of the family and bring together the doctor and the various welfare agencies for which she is a go-between? Criticism of the Public Health Nurse has been free, but even if deserved in one case, I believe praise due her for the remaining ninety-nine. We doctors, like these nurses, should feel resting upon us the care of the patient's family just as vividly as the care of the patient. We should train, direct and incite the nurse to exercise greater initiative in spreading knowledge of sound medical practices among our families and in reporting to us the early symptoms of disease. It is up to us to prove that we, the doctors, rather than the nurses are the progressives in Community Medicine. Too often the Public is ahead of us both. Witness Pondville and other agencies for the care and diagnosis of the patient with cancer.

In our casual conversations with patients we should always impart the most modern medical ideas. In this way the patient is reassured, because he perceives that his doctor is alert, and we doctors are helped because we recite and thus make our own what we have just read. Likewise whenever the patient recounts what he has read of new medical lore, we should receive it eagerly, weigh it, and if we don't know about it, say we will find out the truth in the matter and report upon it, and then not forget to report. Always leave in the patient's mind a reputation for open-mindedness and a good memory and a willingness to do something more than was offered at the formal visit.

And in this discussion about the care of the patient and his family where would I be if I did not mention the activities of the Social Service Worker? I take it that her task is largely to become cognizant of the material problems confronting the patient and to adjust them to those of the family after she has delved into its resources. And here she does not stop because her training has widened her sphere of knowledge so that she is aware of the assistance which can be furnished by state, county, city, town, hospital or by a multitude of charitable agencies. You don't need to preach that the care of the patient includes that of the family to a Social Service Worker.

*Coöperation Between Doctors.* With the passing of the doctor who did all kinds of medicine and surgery, the specialists have multiplied and I believe, contrary to the opinion of many, much to the advantage of the patient. The trouble about specialists has come about not because they know more than the rest of us, but because of lack of complete coöperation among all the medical attendants of the patient. Each of us physicians must be on our guard to work in the closest harmony with all the other doctors of the patient and his family. If we ourselves are ill, we want a specialist. With the woes of our beloved, we fly to the specialist instantly. Therefore, we cannot be too careful to recognize that our patients feel the same way and be ready to propose seeking special advice for those intrusted to our care. Coöperation cannot be one-sided. We simply all must work together and consider our patients with the eyes of the other doctor. Our success in this regard will surely show our own character and we might just as well take this to heart. Such medical interchanges are best fostered in hospitals where doctors doing all sorts of medical and surgical work are closely associated and learn to become

almost absolutely dependent on the special skill of one another. Therefore you can easily understand why I see in the Hospital Family the care of the patient and his family at its best.

*The Hospital Family.* In the care of the patient and his family and in mutual contacts of helpfulness between his physicians I conceive of the Hospital Family as the unit. The hospital patients, of course, form the center of the unit, but in it are just as surely embraced those active in its multiform administrative capacities from the Superintendent to the veriest scrub woman on the stairs, the nursing staff, both graduate and undergraduate, the technicians and social workers, the various medical staffs, and two other groups which have not yet generally received proper recognition, namely the families of the patients and the doctors of those families. The interests of all these groups are intertwined and real progress can only be achieved when there is coöperation between all.

The education of the patient in regard to his disease is increasingly engaging the attention of us all. This education is a function of the Hospital Family. The custom began with tuberculosis. The consumptive patient is seldom cured and his disease is only in part arrested while he is in the hospital, but he learns how it can be arrested, and is taught how to protect his relations from tuberculosis. Then comes the education of the diabetic. He really is expected to study diabetes, to attend classes and to receive individual instruction. More and more are we seeing educational methods proposed and undertaken for patients with other diseases such as rheumatism, anemia and those of the gastrointestinal tract. Then there are other varieties of education which are so much a matter of course that one almost forgets to mention prenatal care and the care of the child. Everywhere today we see education in medicine and to a large degree this is education for the patient's family as much as for the patient and in general it radiates from a hospital because here it can be organized. In a doctor's waiting room there is education to be had, but it is not often enough made alluring.

The education of the patient in the hospital is sure to increase. There is less acute illness and more and more chronic illness. The patient is far more comfortable, and has far less pain. He has much leisure time and recognizes or should be made to recognize that hospital hours must not be wasted and that he should obtain medical knowledge for himself and his family although confined to bed. In all this education I believe the family should share. The patient is nearly always dependent upon the family and the relatives should be shown for the patient's sake what his treatment should be and how he can be protected, and also for their own sakes they should be taught the most modern of medical ideas through which they can avoid what has befallen their relation. They are almost more vitally concerned than the patient, because they probably must bear the brunt of the cost of medical care. For a chronic disease this far exceeds the expenses incident to acute disease although it is to a large extent unappreciated. If the little toe of a diabetic is burned with a heater of most any description, the economic loss is almost five hundred dollars and that without compensation of physician or surgeon.

Education in cancer has been consistently pressed these last few years, but I cannot help feeling that excellent as has been the endeavor, it has in part been misdirected, because it has not sufficiently taken account of the willingness of the patient's family to be educated in cancer prophylaxis. Quite apart from the question of heredity I believe that the best results in the prevention and early detection of cancer should come from the education of the family in which there has been cancer. The family which has had a cancer member should be far more apt and alert to discover a new case than a family in which this disease has not occurred. If cancer in one member of a family was discovered late, that family will be all the more anxious to detect it early, should it appear again, and if treatment in a member was successful that family will see the advantage of prompt recognition. Relatives of cancer patients or better the patients themselves should be encouraged to speak of their recovery and to be proud that their family was bright enough to secure for them prompt treatment. I should like to see the average duration of time computed between the onset and recognition of cancer in families in which cancer has and has not been previously known to exist. Concentrate on the education of the relatives of cancer patients. Use these families to secure further cases at an earlier stage and exploit the reasons for the successful results.

Patients, particularly diabetics, should be taught the educational value and the duty of living lives which will be useful to those similarly afflicted. For years I have tried to impress children with the fact that every honest diabetic day that a diabetic child lives holds out hope to a patient more seriously diseased and have sought to cheer poor old decrepit but courageous diabetic souls, while recovering in an open ward from gangrene, that it was from such as they that came the first proof on a large scale that faithful and prolonged diabetic treatment pays. No diabetic patient can live unto himself alone. His influence, and he must be made to feel this, is never indifferent, but always for good or ill upon other patients.

Disease Consciousness has its advantages as well as its disadvantages. It is always well to face the truth and if disease is present in a family, the family should realize it. It is not

enough that they should feel their duty done if they simply care for the diseased member; the family should take sufficient interest in the disease to join with the doctor and scientist in overcoming it. They should be so educated as to want an investigation after death so as to help the living. And don't forget that an operation during life is attended with pain and for the benefit of the individual, but an operation after death is painless and for the benefit of the family and mankind. I suppose that a large percentage of the funds given for the investigation of special diseases comes from families who have felt the effects of the disease.

May I develop the hospital family idea further? It includes the nurses. To them we doctors owe a great responsibility. Upon them our efforts for our patients depend for execution. If the nurse fails us, we acknowledge that the case is temporarily lost. I think we doctors owe them more and particularly more in the way of health. I realize we take better care of nurses than formerly, but I believe more should be done for them. We should include an x-ray of the chest in their yearly physical examinations. We should make it easy for these girls coming from all over the country to a strange city to secure aggressive attention for their teeth, their throats, their appendices and their feet, and for their postures too. They are in the Hospital Family and it is only justice that everyone in the family should have good health, but should have extra good health. What group of people receiving intensive training have a lower expectancy of years of employment than do nurses?

Are not doctors themselves and doctors' families a part of the Hospital Family? I certainly think they are and that we should develop means by which they not only share in its expenses, but also in its benefits. If we begin at home and develop ways and means by which the not inconsiderable group of the medical fraternity in the community can receive protection against illness and direct aids to better health, it will open up avenues by which other groups may receive like benefits. No one will find fault if we doctors experiment with ourselves. If we can devise hospital insurance for the administrative, nursing and medical staff of a hospital and prove it to be practicable we will have accomplished a good deal for community medicine.

The non-professional employees are a part of my Hospital Family. Do we give them adequate medical attention? Do they really get as much as is offered by various industrial establishments? I doubt it. Through care in selection and promptness in treatment, the sanitarium for tuberculosis for employees of the Metropolitan Life Insurance Company in New York City is now only half-filled with tubercular patients. We hear of the high incidence of tuberculosis among nurses, medical students and young internes, but who has investigated the hospital personnel? Are we not our brother's keeper? An assurance of an interest in their health should make a hospital position especially desirable.

Tuberculosis has been mentioned in our care of the patient and in our care of the Hospital Family, but I would say a word further. In our detection of the disease we are lax, inefficient, and inexcusably late in diagnosis. Notably is this also true for diabetic patients among whom the disease is increasing. This increase among diabetics is explained because the patients live longer and thus are more exposed to tuberculosis; they participate more actively in life and are less sheltered in their homes. Tuberculosis is two to three times more common in diabetics than in non-diabetics, ten times as frequent in diabetic children; of the cases recovering from diabetic coma eight per cent develop it within three years, and my colleague, Dr. Howard F. Root, to whom I am indebted for this information, finds no incipient active case of tuberculosis in a diabetic reported in the literature and only rarely mentioned as having been seen. Even in our own group of 245 cases, despite our recent intensive search, there were but 4 per cent of incipient patients in contrast to the 96 per cent who were well advanced. Tuberculosis can be discovered early if we will use the x-ray method and it is high time that we appreciated this and took steps to employ it more freely. It is ridiculous that we have not profited more from this means, employed so extensively in studying children in the public schools.

Hitherto the expense of an x-ray has barred its usefulness. But I remember the time when a Wassermann test cost $10.00 and rightly so, because few were done and the work was largely experimental. The cost of a blood sugar determination not long ago was $5.00, even $10.00 and also very likely rightly so, although I feel sure any laboratory technician worth her salt would rather do twenty blood sugar tests at 50 cents apiece than two for five dollars each, and indeed upon the former basis a hospital laboratory will profit. Wholesale tests are cheap while retail tests are costly. By the same reasoning I think we should purchase our x-rays wholesale, when dealing with large groups such as nurses, students, doctors, hospital patients, and particularly this should be done for diabetic patients. Doctors don't like to own that a machine is better than their senses, but it is often true and we must concede that these outside aids in diagnosis are vastly better than we can ever hope to attain without them. Let us rejoice that there are such reactions as the Wassermann and the Widal and take advantage of these diagnostic sureties and go on to something else. You and I don't know how to perform these tests, but I don't understand the intricate mechanism of my Ford car, but that car serves me every day and I only hope that my friends have

one as good. It is all right to teach auscultation and percussion, but if we really wish our students to diagnose tuberculosis early, we are not honest unless we tell them to get an x-ray picture.

The expense of taking an x-ray plate will not be prohibitive if we create a sufficient demand so that the films can be procured on a large scale. I am told by the Roentgen-Ray Department of a hospital in Massachusetts that an x-ray (11 x 14) film including the material used in developing it and the service of a technician, but not the overhead of space, heat, light, water and correspondence, is 79 cents, and that one technician can take and develop 34 such films daily. In another state a Roentgen-Ray Department advises me that if 1000 films were purchased, a fair charge would be two dollars apiece and in still another state the Tuberculosis Society purchases such at a local hospital at this price. I believe that it would be to the advantage of our x-ray friends to promote the use of the plain x-ray plate wholesale and to urge us doctors to secure these by hundreds and thousands instead of singly. Unquestionably if this were done it would lead to the discovery of many unknown cases of early tuberculosis. I know my x-ray friends will gladly take a plate for nothing, but no one can ask them to take as many plates as we ought to have.

Now that it has been forced upon me that my diabetics are two to three times as liable to tuberculosis as non-diabetics, that diabetic children are ten times as likely to have the disease as non-diabetic children and that eight per cent of our cases saved with such difficulty from diabetic coma will develop consumption within three years, I shall meet the challenge. I shall do my best to make possible so early a diagnosis of tuberculosis in diabetes as to reverse the picture and thus prove that this special group of 400,000 patients by reason of their being under constant supervision can escape this menace or at least have it discovered at an earlier stage than as if they were nondiabetic. Think of it! We have free examinations to detect the earlier stages of typhoid fever, of diphtheria, of syphilis, but when it comes to the detection of the earliest stages of the major communicable disease which afflicts Massachusetts, we are at sea or forced to beg for charity.

Routine x-rays of the teeth yearly for individuals above the age of fifty are most helpful. The expense of such x-rays is considerable. I believe that if an offer were made to the members of the entire Hospital Family to take such x-rays provided there was application for 500 or more a year, i.e., to allow quantity production, both the hospital and the members would benefit.

In all that is said about x-ray examinations I hope I am making it clear that I would increase the demand for x-ray examinations so much even though the rates per unit were less that in the end those engaged in x-ray work would benefit.

The hospital diabetic family wants a vacation and today any hospital diabetic family which has not an opportunity to allow its members or at least the children to spend two to four weeks at a diabetic camp in the country is behind the times. If the state and the community supply preventoria for tuberculosis, why should they not make possible preventoria for diabetic children who, as I have said, are peculiarly susceptible to tuberculosis?

The hospital patient is usually a temporary patient, a patient lent to the institution and the staff. Loans require interest and repayment. This leads to consideration of the family doctor and his relation to the Hospital Family. Frankly the hospital patients are his children and should be regarded as such. The hospital may help them somewhat, it may educate them a good deal, but unless it reports the methods employed in their treatment, returns them to his care, it is not paying its debts. I know this holds true for diabetes and we certainly try to inculcate in the minds of patients to stick to their family doctors. The patient who develops coma is usually the one who has not visited his family doctor for a long time; the patient who has frequent insulin reactions usually has not reported the same to his physician and it is the uncontrolled, poorly treated diabetic child who is ten times more liable to cataracts and arteriosclerosis than the controlled and carefully treated diabetic boy or girl.

Our present hospital contacts with the family doctor are unsatisfactory. The hospital should consider the family doctor of each patient it admits as an actual member of the Hospital Family. It should place at the disposal of that doctor ready access to its manifold facilities, its laboratories of chemistry and pathology, examinations by x-ray and the electrocardiogram and metabolism, and make these available for emergency tests on holidays, Sundays and even nights and in this manner should put the family doctor to a large degree on a par with the hospital doctor. The hospital should provide for the doctors, who lend it patients, nursing service from its tested graduates by the hour, day or week, including specialized nursing service such as that of the wandering diabetic nurse to assist him in his diabetic families. The hospital, and I know this is being done more and more, should cultivate the interest of the family doctor and secure his coöperation during the actual care of his patients while in wards or private rooms and then return them with such constructive advice that he can take advantage of it in the care of similar cases who wish to remain in their homes. By such measures the family physician should be made to feel that he is entitled to special privileges in the hospital which receives his patient and in fact that he,

too, is really a part of the Hospital Family. Then and then only will our patient and his family receive adequate care.

These are some of the ways in which the care of the patient will include the care of his family and the coöperation of all his doctors. It is one answer to the Costs of Medical Care. To me it furnishes the attractive answer because it provides for all sorts and conditions of men from the poorest to the richest and allows charity to be personal. I realize my Hospital Family does not reach all classes, but it does reach a good many for whom large institutional hospitals do not and ought not to provide. By its very limitations it may succeed. Suppose there are 200 nurses in one hospital, 200 employees, 100 doctors, 500 affiliated doctors and the patients number 5000 annually. All together we have 6,000 individuals representing as many homes and thus reaching out to perhaps 30,000 lives, surely a sufficient number. I conceive of the ideal Hospital Family as touching them all and that if it does not do so, it is failing in its task.

## THE TREATMENT OF ACUTE POLIOMYELITIS WITH THE RESPIRATOR*

BY NEIL LOUIS CRONE, M.D.†

THE Drinker respirator was used at the Massachusetts General Hospital throughout the epidemics of poliomyelitis of 1931 and 1932. A sufficient number of patients have been treated to make it worth while to assess the value of the respirator as a therapeutic agent.

In 1930 there were thirty-two cases of poliomyelitis treated in this hospital. Nine of these were treated in the Drinker respirator. In 1931 there were forty-seven cases, of which fourteen necessitated respirator care. One additional case was treated in 1932. This report, therefore, is based upon twenty-four cases.

There is already growing up a small body of literature which reports in a scientific manner on the use of the respirator, and one series of cases has been studied in detail by Wilson. He has pointed out that poliomyelitis may interfere with the respiratory function in three ways: (1) by paralysis of the intercostal muscles and the diaphragm; (2) by disturbance of the medullary "centres"; and (3) in patients with pharyngeal paralysis, by the collection of mucus or vomitus, either obstructing the respiratory passages directly or leading to irritative spasms of the glottis. In addition we have seen respiration interfered with by the vocal cord obstruction in bilateral laryngeal paralysis.

These three groups overlap clinically. In our experience, also, the cases falling in Wilson's third group are largely of the bulbar type, so that for the purposes of this paper I shall consider our twenty-four cases in three slightly different groups: (1) those with respiratory difficulty due to paralysis of the muscles of respiration; (2) those with respiratory difficulty due to bulbar involvement; and (3) those in which both disturbances are active. Our methods of analyzing these cases and our conclusions are very similar to those of Wilson, although we have not had such good therapeutic results as he.

In the first group, cases with paralysis of the intercostal muscles and diaphragm, the respirator is of greatest value. These cases are often, although not always, of the ascending type, so that by the time the respiratory muscles are involved there has also been extensive and considerable paralysis of the legs and arms. The use of the respirator is justified in these cases on the assumption that after the acute stage is over, the patient will recover sufficient respiratory muscle power to enable him to breathe outside the respirator. A secondary consideration, but an important one from the patient's and the family's point of view, is the hope that sufficient general muscle recovery will occur so that the patient will be able to fill a useful place in society.

Of the cases with paralysis of the muscles of respiration, we have had nine; five have died. The direct cause of death in four cases was cardiac failure and in the fifth hypostatic pneumonia. The last patient was in the respirator with complete paralysis of practically all the body musculature for a period of six months before death. One of those dying from cardiac failure had spent all, or part of, nearly every day for eighteen months in the respirator; she also had complete paralysis of both legs, both arms and the trunk. Roentgen examination disclosed bilateral hydro-ureters with marked dilatation of the renal pelves, which were packed with stones. This complication, probably due to the action of gravity on the ureters as they passed over the pelvic brim, was found postmortem in one other case in this series.

Of the four patients in this group who are living, two have been followed for two years. Both present such paralysis of all four extremities and of the trunk muscles as to keep them bedridden. One is forced to return to a respirator with every upper respiratory infection. Two others of this group have been followed for one

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year. One was in the respirator off and on for seven months and is now practically completely paralyzed and confined to bed. The second, a girl of twenty, has regained some use of the legs and one arm so that she may hope to walk with braces and crutches. She is probably the most hopeful case of the whole series. This is the group from which most is to be expected, and the only one in which we might hope the respirator would serve us well. Yet it will be seen from the observation of the nine cases that the death rate is high and that those whose lives have been saved are invalids.

In the second group, with true bulbar poliomyelitis, there is no paralysis of the muscles of respiration. Just what the cause of respiratory failure in these cases is we do not know. Undoubtedly, however, there is some disruption of function in the bulb which leads to respiratory distress. Many of these patients develop a peculiar inspiratory-gasping type of breathing, as mentioned by Wilson, and this is frequently associated with a pulse of 140-160. Both these signs may be due to bulbar vagal involvement. Some of these bulbar cases retain a considerable amount of mucus in the mouth and throat, of which they are unable to rid themselves, which proves to be a serious complication. Vomiting is a second complication and often the vomitus, being only partially expelled, further plugs and irritates the air passages. A few of the bulbar cases are not of this "wet" type, but these are milder in nature.

One would not expect the Drinker machine to be effective in such cases since its purely mechanical action is of no value in combating the pathological function of the medullary "centres". Many of these patients will not synchronize their respiration with the machine, and even with heavy dosage of morphine to inhibit the respiratory centre, the short, ineffective gasps continue. To combat the dangers from the excess of mucus we have used postural drainage, atropine, and the mechanical sucking apparatus. These give only temporary and partial relief and the patients usually drown in their own secretions. One patient in our series, moreover, developed a bilateral laryngeal paralysis, and an emergency tracheotomy had to be performed.

Since we cannot be certain what the mechanism is in patients of this second group, and since we have no other treatment, we have tried the Drinker respirator in all cases of pure bulbar poliomyelitis with respiratory embarrassment. From the point of view of after-life this should be the most worth-while group, since if there is recovery, there is no distressing crippling or invalidism. Ten patients have been treated in the past three years. None recovered, nor was life prolonged beyond the acute stage in any of them. They were in the respirator for varying intervals, the shortest time being one hour and the longest seventy-two hours. All were of the "wet" type.

As the result of the above study, we have come to regard certain signs as of grave prognostic significance in cases of bulbar poliomyelitis: (1) vomiting after the prodromal period; (2) rapid pulse; (3) retained mucus in the throat; and (4) gasping, inspiratory efforts. A further sign, which needs confirmation, is the persistence of excessive amounts of sugar in the spinal fluid.

The exact cause of death in bulbar poliomyelitis has not been determined. Clinically, the patients remain cyanotic in the respirator, even when oxygen and carbon dioxide are administered. Anoxemia is, therefore, always present. The rapid rate of the heart also leads to circulatory failure and possibly to inefficient circulation in the lungs and medulla itself, so that a vicious circle is established. A further cause for embarrassment of the respiratory function is edema of the lungs, a finding often confirmed at necropsy. Irrationality and delirium, out of proportion to the temperature, were frequently observed.

In this group then, experience bears out our theoretical considerations. None of these ten patients have survived, nor has life been significantly prolonged by the use of the Drinker respirator.

The third group are either cases of the ascending type with involvement of both the respiratory muscles and the bulbar centre, or of the descending type, with similar involvement. Some patients have started with paralysis of the intercostal muscles and the process has then migrated both up and down the central nervous system. As a means of treatment, the respirator is effective in inverse proportion to the amount of bulbar involvement. The complications presented are those of the first two groups.

Five such patients have been treated in the respirator. Three of these patients died, two have survived. One of those who survived had, on entry, a bilateral facial paralysis as well as total paralysis of both arms and legs. Shortly after admission the child became cyanotic, was placed in a respirator, and in a few days could be out for a short while at a time. At no time was there any involvement of the cranial nerves other than the facials; excessive mucus, rapid pulse, vomiting, or gasping respiration were not observed. It is presumed that the respiratory difficulty in this case was due, entirely or for the most part, to intercostal and diaphragmatic paralysis. This child left the hospital with practically complete paralysis of both legs but is now, two years later, able to be about with braces.

The second survivor in this group is a case of only two and one-half months' duration. When first seen, there was extensive paralysis of the arms, legs and trunk as well as of the intercostals and diaphragm, diplopia and slight temporary difficulty in swallowing. There were no other cranial nerve palsies at any time and this patient likewise did not show the unfavorable bulbar signs noted above. It is probable here also that the respiratory difficulty was due to paresis of the intercostal muscles and diaphragm rather than to bulbar respiratory involvement. At present, the patient is still in the respirator all of the night and two hours during the day. There is still extensive paralysis of the arms, legs and trunk muscles.

Of the three cases in the third group who died, two were of the ascending and one of the descending type. All died of the bulbar disturbances mentioned above.

The age distribution of the whole twenty-four cases is quite wide. The youngest patient was five years old; the oldest, thirty-six. Eight were under fourteen years of age; of these four have survived. Sixteen of the patients were fourteen or over and of these, three are living and thirteen are dead.

## CONCLUSIONS

1. A group of twenty-four patients with acute poliomyelitis treated in the Drinker respirator has been reviewed.

2. These twenty-four cases are divided into three groups, dependent on the primary cause of respiratory failure: (1) involvement of the respiratory muscles; (2) involvement of the bulbar "centres"; (3) involvement of both mechanisms.

3. In the first group, patients with respiratory muscle involvement, there were nine cases. Of these five died and four survive. Two of the four survivors have been followed for two years and are invalids confined to bed. The other two have been followed for one year; one of these is completely paralyzed in legs, arms, and trunk, and confined to bed; the other shows sufficient recovery to justify the hope that she may walk with the aid of braces and crutches. This is the group in which the respirator could, theoretically, be expected to be of most value.

4. In the second group, patients with bulbar involvement, there were ten cases. All of these were fatal. The possible causes of death are discussed and reasons are brought forward to demonstrate that the respirator may not be expected to aid in such cases. In the bulbar cases the most unfavorable signs are the following: (1) a rapid pulse; (2) excessive mucus; (3) vomiting after the prodromal period; (4) short, gasping inspiratory efforts; and (5) persistence of a high spinal fluid sugar.

5. In the third group there are five cases. Of these, three were fatal and two survived. One of the survivors had little or no involvement of his medulla but presented a nearly complete paralysis of arms and legs. He is on braces now, after two years, with almost no use of his arms. The second survivor has been followed for only two and one-half months. He also had little and temporary involvement of cranial nerves. He has, at present, an extensive paralysis of arms, legs and trunk and spends all night and two hours during the day in the machine. It is felt that respiratory difficulty in both these cases was due to intercostal and diaphragmatic paresis and not to bulbar involvement.

6. Of twenty-four patients treated with the Drinker respirator, eighteen are dead and six are living. Of the six who survived, some will be permanently paralyzed.

7. Two patients who were in the respirator for the longest periods developed extreme dilatation of the ureters and renal pelves, probably as a result of the action of gravity on the ureters as they pass over the pelvic brim.

8. It is felt that the use of the respirator is always justified in poliomyelitis for symptomatic relief, and that most is to be hoped for in those cases with intercostal and diaphragmatic paralysis. The most unfavorable type is that in which the respiratory difficulty is due to bulbar involvement.

## REFERENCES

1 Drinker, Philip, and McKhann, Charles F.: The use of a new apparatus for the prolonged administration of artificial respiration. 1. A fatal case of poliomyelitis. J. A. M. A. 92: 1658 (May 18) 1929.

2 Wilson, James L.: Types of respiratory failure in poliomyelitis. New Eng. J. Med. 205: 597 (Sept. 17) 1931.

3 Wilson, James L.: Acute anterior poliomyelitis. Treatment of bulbar and high spinal types. New Eng. J. Med. 206: 887 (April 28) 1932.

4 Wilson, James L.: Treatment for respiratory paralysis in poliomyelitis with the Drinker respirator. Tr. Am. Pediat. Soc. 43: 35, 1931.

# HERNIATION OF THE FUNDUS OF THE STOMACH THROUGH THE ESOPHAGEAL HIATUS: WITH SPECIAL REFERENCE TO ITS ROENTGENOLOGIC DIAGNOSIS

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## INTRODUCTION

DIAPHRAGMATIC hernia is one of the hidden deformities which has always attracted considerable attention. As a result of this interest a large literature has accumulated, dealing with the embryology, anatomy, classification, differential diagnosis, roentgenologic study and treatment of the condition. In this paper we propose to consider only the esophageal orifice hernias, paying particular attention to their roentgenologic study. As here used the term, esophageal orifice hernia, applies to a protrusion of a portion of the fundus of the stomach through the esophageal hiatus of the diaphragm into the thorax. This condition is far from common and not until the roentgen ray became generally used was it possible to make a definite diagnosis during life. The question is pertinent: Is it as rare a condition as it was formerly thought to be?

Ambroise Paré[1] and Jean Petit[2] are both credited with being the first to describe a diaphragmatic hernia. In 1575 Paré reported two cases of traumatic herniae, and in the 1844 edition of Petit's "Malaise Des Os Oeuvres Complete" we find reference to two cases, one traumatic and one congenital. In 1769 Morgagni[3] prepared a monograph on diaphragmatic hernia and mentioned herniation through the esophageal hiatus. Bowditch[4] in 1853 collected 88 cases from the literature and Lacher[5] in 1880 tabulated 117 congenital and 150 traumatic cases.

Since the advent of the roentgen ray at the beginning of the twentieth century, and especially since the use of the opaque meal in 1905, cases have accumulated with greater rapidity. In 1911 Eppinger[6] tabulated 653 cases of hernia through the various parts of the diaphragm, eleven of which involved the esophageal orifice. The next year Giffin[7] summarized the literature and found that, of 650 cases reported, only 15 were diagnosed during the life of the patient. Breitner[8] found that of the large number of cases reported up to 1921 only 45 were diagnosed during life. Richards[9] tabulated 137 cases from 1900 to 1923 and found that, of these, 23 were through the eosphageal orifice.

The literature of the past decade indicates that herniation of the fundus through the esophageal hiatus is more common than is supposed. Thus in 1924 Carman and Fineman[10] reported 12 cases, six of which involved only varying portions of the fundus of the stomach. L. B. Morrison[11] in 1922 reported 12 cases of congenital hernia of a portion of the fundus. In 1925[12] this number was increased to 42 cases out of 3,500 gastric cases studied and the next year Healy[13] reported that this series had been further increased to 53. Up to date they have observed 314 cases of herniation of the fundus of the stomach[14]. In 1926 Pancoast and Boles[15] reported 20 cases from the Mayo Clinic, 15 of which were found in 1923-1924. In 1925 Hedblom[16] analyzed 378 cases from the literature. Of this number 163 cases were described as to location, twenty per cent of which were esophageal hernias. Akerlund[17] reported 30 cases of hiatal hernias in 1926 and found 60 others in the literature. Truesdale[18] has reported numerous cases of diaphragmatic hernia with operative treatment during the past decade. Greenwald and Steiner[19] found 82 cases of diaphragmatic hernia in childhood reported in the literature between 1912 and 1923. In 1929 Ude and Rigler[20] reported 19 cases of hernia through the esophageal orifice. They are of the opinion that this is the most frequent site of diaphragmatic hernia. In 1930 Ritvo[21] reported 60 cases of esophageal hiatus hernias which he had observed over a period of five years. In 1931 Hedblom[22] again summarized the literature and tabulated 1435 cases. Of this number 190 were exclusively esophageal hiatus hernias diagnosed roentgenologically. He points out that careful roentgenologic examination has revealed a relatively large number of small hernias. If the cases so revealed by roentgenologists were added to those which he tabulated "the esophageal hiatus hernias would represent over two-thirds of the non-traumatic type and over one-third of all the reported cases." Harrington[23] is of the opinion that hernia of the fundus of the stomach through the esophageal orifice is the most frequent of the various types of diaphragmatic hernia.

## EMBRYOLOGY

The anlage of the pericardial, pleural and peritoneal cavities, the embryonic coelom, is formed by the end of the fourth week as two parallel canals on either side of the midline by the coalescence of clefts in the mesoderm. These two canals soon unite at the anterior extremity of the embryo to form a horseshoe-shaped pericardial cavity[24]. Dorsal to this cavity lies the foregut, which has invaginated while the cavities were forming, and ventrally it is closed off by the infolding and fusion of the splanchnopleure. Coincidental with the descent of the

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primitive diaphragm and the increasing flexion of the embryo, the pericardial cavity comes to lie nearly at right angles to the plane of the paired pleuroperitoneal cavities and communicates with them dorsocaudally. These paired cavities are separated from one another longitudinally by the primitive mediastinum and the mesentery. With the caudal extension of the gut and the disappearance of the caudal portion of the ventral mesentery, the peritoneal portion of the parallel canals unite to form the common peritoneal cavity. The pleuroperitoneal cavities at first communicate laterally with the extra-embryonic coelom, but by the formation of the common peritoneal cavity and the infolding of the splanchnopleure on each side they become entirely separated, leaving the umbilicus as the last point of closure.

The division of the coelom into separate cavities is begun by the fourth week. This is accomplished by the following membranes: (1) the dorsal mesentery, (2) the septum transversum, (3) the paired pleuropericardial membrane, and (4) the paired pleuroperitoneal membranes.

The septum transversum (His) arises during the third week as a mass of mesodermal cells along the walls of the vitelline veins as they pass in the ventral mesentery to the heart, paralleling the foregut. These vessels bulge laterally into the coelom, and, with progressive enlargement, finally meet and fuse with the somatic mesoderm. This forms a transverse partition caudal to the heart, separating the pericardial and the peritoneal cavities ventral to the gut. Dorsally the septum is incomplete and here a communication exists on each side between the pericardial and the pleuroperitoneal cavities. These passages are known as the pleuroperitoneal canals, in which lung buds develop. The septum transversum is early differentiated into two portions, a cephalic layer which is the anlage of a large part of the diaphragm and the floor of the pericardial cavity, and a caudal layer into which the liver penetrates.

In the 2 mm. embryo the septum transversum lies in the cervical region, occupying a transverse position. It then migrates caudally, reaching its final location opposite the first lumbar segment in the second month.

The complete separation of the pericardial, pleural and peritoneal cavities is accomplished by the formation of the pleuropericardial and pleuroperitoneal membranes. The chief structures concerned in this process are the common cardinal veins (ducts of Cuvier), which lie in the lateral walls. They curve around the pleuroperitoneal canals, passing from the dorsolateral walls and bulge into the canals. This constriction is called by Mall the pulmonary ridge. The anterior portion of the ridge forms the pleuropericardial membrane and later separates the pleural and pericardial cavities. Its caudal portion constitutes the pleuroperitoneal membrane and separates dorsolaterally the pleural and peritoneal cavities[9]. The dorsal mesentery contributes the dorsomedial part of the diaphragm including the area through which the esophagus and vena cava pass. The failure of the pleuroperitoneal membrane to close, results in the herniation of abdominal viscera at this point[25]. After its formation the pleuroperitoneal membrane lies anterior to a portion of the lung and encircles it. The dorsal end of the septum transversum descends more rapidly at this time than its ventral portion, thus bringing the pleuroperitoneal membrane to a position caudal to the lung. The pleuropericardial membrane is held behind during this migration by the phrenic nerve which passes through it. The lung now occupies a cone-shaped cavity between the pleuropericardial and pleuroperitoneal membranes. The further migration of the septum transversum brings these structures to lie at right angles to one another. The final closure of the pleuroperitoneal and pleuropericardial membranes occurs at the 19 to 20 mm. stage. The right pleuroperitoneal canal closes first due to the presence of the right lobe of the liver.

The primitive diaphragm thus formed is made up of mesenchymal substance. Its muscle elements originate in a pair of cervical myomeres at the fifth cervical segment. They grow in from the lateral wall, aided by the pressure of the expanding lungs. The distribution of the muscle tissue when complete is uniform throughout. The central tendinous portion results from atrophy of the muscle in this area.

The development of the omental bursa is another embryological factor of significance in the etiology of esophageal hiatus hernias. The structure appears in the 3 mm. embryo as a finger-like projection of the peritoneal cavity into the dorsal mesentery on either side of the esophagus. The projection on the left is normally obliterated at an early stage but the one on the right persists for some time and eventually gives rise to the omental bursa. Normally this is constricted off at the esophagogastric junction after the descent of the stomach into the peritoneal cavity and is obliterated. It may persist at the side of the esophagus and thus form a likely spot for herniation of the stomach.

The stomach appears in embryos of 4 to 5 mm. as a lateral fusiform enlargement of the foregut caudal to the lung anlages[24]. It grows slowly at first and remains above the septum transversum up to the 11 to 12 mm. stage. It then moves caudally with the rapid elongation of the esophagus so that it lies caudal to the diaphragm by the time of its closure in the 19 to 20 mm. embryo. The stomach is attached to the dorsal wall by its dorsal or greater omentum and to the liver by its ventral or lesser omentum. The rapid growth of the dorsal border produces the fundus, the characteristic les-

ser and greater curvatures and the rotation to the right on the long axis.

Should there be any retardation in the growth of the esophagus, any portion of the stomach that may have failed to reach its normal position at the time of the closure of the diaphragm will be retained in the thoracic cavity.

### CLASSIFICATION OF ESOPHAGEAL HIATUS HERNIAS

In 1922 L. B. Morrison[11] reported before the New England Roentgen Ray Society the first twelve of his series of cases which he termed "congenital hernia of a portion of the fundus of the stomach through the esophageal hiatus," and at that time made reference to the short esophageal type of hernia. In 1926 Akerlund[17] published a paper on this subject based on the roentgenologic examination of 30 cases. He proposed that this condition of herniation of the fundus through the esophageal hiatus be called "diaphragmatic hernia of the esophageal hiatus" or "hiatal hernias". He differentiated all such hernias into three groups on an anatomical basis, among which there are certain transitional forms. His classification is as follows:

1. Hiatus hernias with congenitally short esophagus.
2. Paraesophageal hiatus hernias in which the esophagus is not short and is not herniated.
3. Other hiatus hernias in which the esophagus is not shortened, but in which the distal portion of the esophagus is a part of the hernial contents.

Of these hernias the last group is the most common while the short esophageal type is the most rare.

### ETIOLOGY AND PATHOLOGY

In the hiatus hernia with shortened esophagus the distal portion of the esophagus does not reach the level of the hiatus of the diaphragm. Consequently a varying amount of the stomach comes to lie in the thorax. In this type, the esophagus ends a little more than a finger-breadth to 7 or 8 cm. above the plane of the diaphragm and the fundus lies within the hiatus. When the major portion or all of the stomach is in the thorax, the condition is called "Thoracic Stomach." Bailey[26] in 1919 is usually credited with first describing this condition, but Bright[27] reported a case from Guy's Hospital in 1836, and Bund[28] reported a case in 1918. We found at least 18 undoubted cases of thoracic stomach in the literature. Bailey states that "there is no evidence that the stomach had herniated into the thorax. Its positions and relations may be explained by supposing that the anlage of the stomach lay abnormally far anterior on the alimentary canal. The peritoneal pocket around the stomach developed before the descent of the diaphragm, and finding the peritoneal relations already developed, the diaphragm would necessarily leave above it, as it descended, not only the stomach but the expansion of the peritoneal cavity."

There are several other opinions concerning the cause of the short esophagus and the abnormal position of the stomach. Tonndorf[29] has suggested that there is an idiopathic failure of the alimentary tract to fix itself at the diaphragmatic anlage thus slowing the descent of the stomach. Bund[28] is of the opinion that this abnormality is due to the delayed closure of the right pneumato-enteric recess (omental bursa) along the esophagus, and that the shortening of the esophagus is secondary.

That this dilated portion of the digestive tract just above the diaphragm in these cases is truly a part of the stomach and not a dilatation or diverticulum of the esophagus has been demonstrated by three methods. Findlay and Brown Kelley[30], reported that they had shown this to be stomach by esophagoscopic examination. They further demonstrated this by an histologic study of a portion of tissue removed from the dilated area during such an esophagoscopic examination. Roentgenologically the difference between the esophagus and the herniated stomach can be seen by the change in the peristaltic movements and the presence of rugae.

Reposition of the herniated stomach into the abdomen is well-nigh impossible since it is held in its abnormal position by the esophagus which is relatively short for that particular patient.

The paraesophageal hernias form a significant group, occurring often in old people, particularly in women. A portion of the stomach passes upward between the esophagus and the enlarged hernial ring, protruding into the thorax. The lower end of the esophagus and the cardia remain below the diaphragm. Strictly speaking, there is normally no "esophageal opening", that is the digestive tract is everywhere intimately enclosed by the dorsal, and above the diaphragm by the ventral mesentery, through which the esophagus passes. The only "opening" is that created by the failure of the diaphragmatic muscle to closely surround the esophagus. The more the muscle fails to do this the greater is the amount of the membranous partition composed of peritoneum, pleura and interstitial tissue, which under certain conditions, may become the site of a hernia. For this reason the esophageal hernias are commonly contained in a sac formed of relaxed membranous tissue. The great majority of them as found in adults, are of the acquired type and result from repeated increase in intra-abdominal pressure occurring with coughing or straining. This pressure transmitted by the stomach to the hiatus causes a pressure atrophy of the ligamentous and muscular tissue of a hiatal ring, which, perhaps, is already abnormally large or weakened, thus causing further enlargement.

Then, too, there are the congenital cases of

this condition. Here again Bund's[28] theory may be applied. If for any reason the infracardiac bursa, which at an early stage communicates with the omental bursa and hence with the general abdominal cavity, should persist and remain open below, there would then be formed a passage beside the esophagus and through the diaphragm. This would afford an extremely vulnerable spot for a viscus to force its way upward along the esophagus, through the muscular ring, simulating in every way a general dilatation of the opening.

Since the esophagus is of normal length with its distal end in the abdomen, these cases are amenable to surgery provided no adhesions have formed and the enlarged orifice can be sutured together.

Between this group of cases and the third type of hiatal hernias there are many transitional forms. This last group of hiatal hernias are those in which no short esophagus exists and which are not paraesophageal. The lower end of the esophagus and the fundus of the stomach have both passed up through the enlarged hiatus as part of the hernia. The esophagus is relaxed, may become tortuous and forms no hindrance for replacement of the hernia, provided no adhesions have developed. These, the largest of the hernias through the esophageal hiatus, are apt to give the most severe symptoms of hiatal hernias, and include the majority of the operative cases. The etiological factors are the same as for the paraesophageal hernias.

Other abdominal organs in addition to the stomach will herniate through an enlarged esophageal hiatus. The colon and the small intestine are the most frequent offenders. As a point of fact every abdominal organ with the exception of those in the pelvis has been found in the thorax in hernias through various parts of the diaphragm.

## SYMPTOMS

Diaphragmatic hernia is surprisingly variable in its symptomatic manifestations. There is no one characteristic symptom or syndrome which points unmistakably to such a lesion. The complaints of the patient depend chiefly upon the organs involved in the hernia, the extent to which their normal function is disturbed, and the degree to which the presence of abdominal viscera in the thorax impairs respiration and circulation. Hernia may exist symptomless for years only to be discovered during a routine roentgenologic examination of the chest or abdomen, at operation for some other cause, or at postmortem examination. Many of the cases reported in the literature have been free from symptoms due directly or indirectly to the hernia.

The symptoms may be referable to the thorax or abdomen, or to both. Of the abdominal manifestations, vague pain or distress just under the ensiform process during or after meals is the most common. Frequently this dull pain starts in front, radiates to the back in the infra- or supra-scapular regions, thus resembling a chronic cholecystitis. Often the pain radiates to the left shoulder. This pain together with the regurgitation of recently digested food, particularly if the patient is in the recumbent position, is the most characteristic symptom of hiatal hernias. Patients soon learn that this is relieved by standing up or walking around. This type of pain together with its remedy is often very suggestive of this condition. Night awakening with abdominal distress, nausea and vomiting of a foamy acid residue occurs. Constipation is often associated with this condition and melena is frequently present. A secondary anemia is not at all uncommon. Some patients complain of difficulty in swallowing, which is probably due to pressure of the herniated portion of the stomach on the esophagus. Gaseous eructations and heartburn are frequent symptoms.

A feeling of fullness in the chest after eating or retiring is the most common thoracic manifestation. Dyspnea, cyanosis, palpitation, and a feeling of suffocation or pain have been reported. Hiccup and cough are occasionally noted.

All of these symptoms, both abdominal and thoracic, are more severe when large quantities of food are taken, and are usually relieved by standing up or by vomiting. On the whole they are usually mild. As the stomach is the only organ herniated in most cases, the symptoms are mostly gastric in type.

## SIGNS

The physical signs vary with the size of the hernia and the position of the herniated organ, since the change in physical findings is chiefly due to the presence of abdominal viscera in the thorax. Patients with long-standing hernias may show only an emaciation due to the inability to take and retain sufficient food. The percussion note may vary from tympany to dullness, depending upon the size and content of the herniation. A small peripheral hernia may produce distinct changes, while a large hernia in the posterior mediastinum may not alter the percussion note. Displacement of the heart has been noted with large hernias. However tympany over the lower central portion of the thorax, changing to dullness when food or drink is taken, is perhaps the most frequent finding, if any signs at all can be demonstrated.

Should a portion of the colon or small intestine become involved in the hernia the symptoms and signs will become more marked. Even the findings of partial or complete obstruction may appear.

## DIAGNOSIS

The variation and vagueness in the symptoms and signs makes a positive clinical diagnosis

well-nigh impossible. It is important to recognize that the symptoms of hernia may resemble closely those of the more common abdominal conditions such as gall-bladder disease, peptic ulcer and carcinoma of the esophagus or stomach. Angina pectoris must also be considered at times. Physical findings may be very similar to those of pleuritis or pneumothorax with or without effusion. It must be borne in mind that a hiatal hernia may exist with other lesions. Thus gall-bladder disease or gastric ulcer have been observed in patients with such hernias.

Each of these conditions which must be considered differentially, usually has its typical history and clinical evidence. It is in the atypical cases that diagnosis is difficult and the possibility of esophageal hernia should be recalled. In any case in which the clinical picture in any way suggests diaphragmatic hernia and ordinary study does not establish the diagnosis, the special roentgenologic examination is indicated. As more cases of this type of hernia are recognized and reported the clinical picture will become more clearly defined and this condition will be recognized as a not uncommon cause of upper abdominal distress.

### ROENTGENOLOGICAL STUDY

Herniation of the fundus through the esophageal hiatus is one of the concealed abnormalities which roentgenological study has exposed. The clinical evidence is often very suggestive but clinical diagnosis is so uncertain that a thorough roentgenological examination is essential for a final diagnosis.

J. M. W. Morison[31] has pointed out that "The number of cases diagnosed apart from x-ray examination is no greater than it was in the eighteenth century, the great majority of cases being discovered in the x-ray room during routine examination of obscure stomach and chest cases, or in the pathology department in the unexpected cases."

The technic of a roentgenological study is exacting and must be carried out with accuracy and care. Roentgenograms are extremely difficult to obtain. Even the most careful technic may fail to show the lesion. This is due to the inability of the patient to remain quiet, the small size and the superimposed shadows of the heart, spine and thoracic cage. The most satisfactory method for obtaining radiograms is to visualize the hernia with the fluoroscope, replace the screen with a film and make the exposure with the patient in exactly the same position. Serial radiograms are occasionally of considerable value.

The roentgenological examination is first made with the patient in the upright position. The anteroposterior and oblique views are obtained. The position and movements of the diaphragm, the cardiac shadow, and the posterior mediastinum are studied. Occasionally a gas bubble may be observed in the oblique position above the diaphragm. LeWald[32] has pointed out that the absence of the usual *magenblase* in the fundus is very suggestive of herniation of the stomach. A fluid level with the gas bubble above it may occasionally be observed in cases in which the fundus and the esophageal opening are above the diaphragm.

A barium meal is then given. There is normally a slight sphincter effect at the cardia occurring with the respiratory movements. The column of fluid is from two to three inches high in the lower esophagus when the barium is rapidly ingested. In the hernia cases the column of fluid may remain four to five inches high. The esophagus seems a trifle dilated. This dilatation does not occur with the short esophageal hernias, but is frequently noted in the other two types of hiatal hernias in which the filled hernia often partially obstructs the esophagus. After the barium has been ingested, an attempt to fill the hernial pouch by deep inspiration and manual pressure on the epigastrium should be made.

Usually the examination in the erect position does not demonstrate the existence of a hiatal hernia unless it is large and partially blocks the esophagus. Consequently if the roentgenological study is limited to this position alone, the majority of the hiatal hernias will be missed. However, one should never fail to examine the esophagus and stomach in the upright position.

The patient is then placed on the fluoroscopic table in the horizontal position. There is no one single position in which it is possible to demonstrate the hernia in every instance. It is essential to study the patient in the prone, supine and oblique positions. Full inspiration and normal pressure on the epigastrium should be used in each position to increase intra-abdominal pressure thus forcing the gastric contents into the fundus, cardia and esophagus. The Trendelenburg position, or even slightly elevating the pelvis with a pillow are important aids.

Menge's position is the one position which is most likely to lead to a diagnosis. In this position the patient is lying face down with the right arm extended along the side of the body, left arm on the pillow, and the left knee drawn up so that the body rotates somewhat obliquely to the left, giving a view of the mediastinal space. Often the hernia cannot be observed on the first examination, but a repetition of the study at a later date reveals a rapidly filling pouch. In such cases the failure to make the diagnosis may be due to spontaneous reduction of the hernia at the time of the first examination, or to the fact that the herniated portion is small or lies behind the cardiac shadow.

A thin barium meal given to the patient in Menge's position passed down rather rapidly with peristalsis only moderately visible. The thick barium passed down more slowly, showing

slight variations in the size of the esophagus. When it reached the diaphragm, in the normal individual, there seems to be a pinchcock effect, that has been so nicely described by Chevalier Jackson[33]. This opens up and allows a bolus of food to pass through the esophageal hiatus, down that portion of the esophagus below the diaphragm and into the stomach approximately one inch below. This abdominal portion of the esophagus may be of variable length, depending upon the shape of the fundus of the stomach. One must carefully observe the cardiac orifice of the esophagus and the size of the hiatus must be watched as the esophagus empties. As the mucosa of the esophagus comes into mutual contact we notice the longitudinal lines where the barium projects into the various folds of the esophagus. These folds are characteristic of the esophagus and have been described by Mosher[34]. The peristaltic waves over the esophagus are typically smooth and even throughout its length.

In the study of the normal fundus of the stomach in relation to the cardia, we find that the esophagus, as seen in the fluoroscope or plate, extends down posterior to that portion of the fundus which lies anterior to the esophageal hiatus. In some instances the esophagus comes down to the right of the stomach and then crosses to the left at an oblique angle and enters the fundus, much as we see in anatomical dissections.

It is observed in certain stomachs which may be described as of the infantile type, where the esophagus barely passes through the diaphragmatic orifice before it empties into the fundus. The esophagus then enters the stomach at its most superior aspect, or a little to the right immediately under the diaphragm, rather than crossing obliquely to the left and entering an inch or more below the orifice. This condition undoubtedly represents a transitional form between the normal stomach and the short esophageal type of hiatal hernia. If the stomach is completely filled with the opaque substance and the patient in the supine position inspires deeply, this portion of the fundus projects up into the hiatus similar to the beginning of the protrusion of the omentum or bowel into an inguinal hernia, so that it acts as a potential dilator of the esophageal orifice.

Now, in following this type of stomach a bit farther, but with a slightly short esophagus, we find occasionally (in watching the bolus of barium pass down the esophagus) a pinch-cock effect one or two centimeters above the diaphragm. There is a rounded bulge just below this constriction in which the peristalsis differs from that of the esophagus and resembles that of the stomach. It is extremely difficult to plate this and the observations are made mostly with the fluoroscope. A careful observation of this rounded area shows the irregular arrangement of the rugae which we see in the normal fundus of the stomach instead of the parallel lines in the esophagus above the bulge. This difference can be visualized only while the sac contains a small amount of barium and is obscured if it is completely filled. The appearance of the rugae is an important observation since it, together with the change in peristalsis, is the roentgenologic proof that the ovoid shadow above the diaphragm is actually a portion of the stomach and not a dilatation or diverticulum of the esophagus. It is interesting to note that in this condition and in the stomach described as infantile that incompetency is a fairly constant observation. It is probably due to a variation of the arrangement of the muscle fibers, although this has not been demonstrated anatomically.

In those cases, in which the esophagus ends three to five centimeters above the diaphragm, a definite portion of the fundus is seen in the thorax. In such a case one may often visualize two slightly constricted areas, between which is a dilatation. The superior constriction occurs at the junction of the esophagus and the stomach, while the inferior one appears at the level at which the stomach passes through the diaphragm. Either of these may be absent or overlooked in the examination. The esophagus above the dilated area is straight and does not appear kinked as if it were relaxed. One is perfectly certain that such an esophagus is not due to contraction because of the long malposition of the herniated fundus since the walls of the esophagus behave in a normal manner. One should carefully study the relations of the esophagus to the hernia in every instance. This is best accomplished by completely filling the stomach and hernia after the movements and the hernia have been visualized. This is important in differentiating the various types of hernia, in observing the reduction of the hernia, and in proving the presence of adhesions.

After making a careful study with the patient in the Menge's position the examination is completed by fluoroscopy with the patient again in the standing position. The purpose of such a procedure is to observe the spontaneous reduction of the hernia under the weight of the barium in the stomach. Thus in the roentgenologic study of the short esophageal hernias the following important factors have been noted:

(1) No abnormality is noted when the patient is standing.

(2) There is a gradual transition in a definite sequence from the patient with a normal stomach, through the infantile type with the esophagus entering at the peak of the fundus, on to the short esophageal hernias in which the relatively short esophagus enters the fundus above the diaphragm.

(3) Above the diaphragm two slightly constricted areas may be observed between which is a dilatation characterized by fundal rugae.

(4) The esophagus is straight.

(5) The herniated portion is never reduced when the patient assumes the upright position.

In the para-esophageal hernias the *magenblase* may appear normal. Usually, however, it is small and pushed to one side. The barium is seen to run down the esophagus and enter the stomach. As the stomach distends, the barium appears around or to one side of the esophagus which is already filled by the opaque substance as it is passing downward. The hernia may appear to the right, left, front or back of the esophagus, usually to the left and posterior. In some cases it may appear to surround the esophagus.

The fluoroscopic study of the third type of hiatal hernia is characteristic enough in typical cases to permit a differential diagnosis. The esophagus is usually seen to be tortuous or relaxed. It enters the fundus above the diaphragm, and the characteristic rugae are seen in the thorax. The spontaneous reduction of the hernia may be seen under the fluoroscope. The tortuosity of the esophagus disappears with such a reduction.

It should be pointed out that roentgenological differentiation of these three groups of hiatal hernias is not an easy task and is uncertain. It may be considered that such a differentiation is only of academic importance. However, a diagnosis is possible in typical cases and should be made in order for the roentgenologist to offer recommendations for the type of treatment to be followed.

Roentgenologic differential diagnosis of hiatal hernias includes diaphragmatic hernia of other types, eventration, absence of the left half of the diaphragm[35], esophageal and gastric diverticula, cardiospasm, cardio-esophageal relaxation and hour-glass stomach.

Diaphragmatic hernia of other types than the hiatus hernias may involve any portion of the diaphragm and include other organs than the stomach. The site of the hernial opening, the relation to the esophagus and organs involved may be demonstrated by the barium meal or enema. Differentiation of eventration and hiatus hernias depends on the visualization of abdominal organs below an elevated dome of the diaphragm. Kirklin[36] points out that in eventration the upper level of the stomach contents is at the hiatus, while in hernia of the stomach it is apt to be above it. "Absence of the left half of the diaphragm may be best noted in a lateral examination. In this position in the normal person, both the right and the left leaves of the diaphragm can be seen throughout their entire extent; hence, the absence of one leaf or a portion of it can be readily determined. In eventration of the diaphragm, no matter how thin or how high the outline of the diaphragm, it can be determined"[34].

Diverticulum of the esophagus always lies in contact with the esophagus. It fills directly from the esophagus, lies well above the diaphragm and is best seen when the patient is erect. A hernia fills from the stomach, is in contact with the diaphragm, is seldom seen except in the horizontal position and gastric rugae may be demonstrated. A diverticulum of the stomach is below the level of the diaphragm, fills from the stomach in the upright position and retention of opaque material is noted after the stomach has emptied.

Cardiospasm is characterized roentgenologically by a high column of barium in the esophagus, and a dilatation of the esophagus above a constricted area. In cardio-esophageal relaxation the barium descends quickly into the stomach, and is seen to run back when the patient is recumbent. No hernial sac above the diaphragm can be visualized in either of these conditions.

Hour-glass stomach may be distinguished by the fact that it lies below the diaphragm and the media enters the upper portion of the stomach first while in hiatus hernia the barium enters the lower intra-abdominal portion first.

## TREATMENT

The treatment of esophageal hiatus hernias may be either medical or surgical. The decision as to which of these resources is to be followed rests upon two factors, the severity of the symptoms and the roentgenological findings. Should the roentgenological studies reveal that the hernia is of the short esophageal type, surgical intervention, retraction of the stomach and suture of the diaphragm will probably be unsuccessful. The roentgenologist may further be of assistance by observing the size of the hernia, the symptoms when the hernial sac is filled, and the amount of dilatation of the esophagus. He can also suggest the site of operation, whether the approach should be thoracic or abdominal, by determining the position of the diaphragm, the width of the angle at the ensiform cartilage and the extent of ossification seen in the costochondral junction.

Medical treatment is entirely symptomatic and varies with the individual case. It consists largely in preventive measures and dietary management. The patient should be directed to avoid all conditions which increase intra-abdominal pressure such as vomiting, straining at stool, sudden severe exertion, or tight abdominal bands. The avoidance of overdistention of the stomach with large amounts of food or fluid is important. Gaseous distention and constipation should be remedied. Patients should be put on a dietary régime consisting of frequent small meals of high caloric value. The problem here is to maintain nutrition and to avoid having large amounts of food in the stomach at any one time. Cabot[37], Crohn[38], and Schisler[40], have reported cases which have been successfully treated by strict diet. Any variation from the diet resulted in return of symptoms. The patient should not lie down immediately after eating as this is apt to cause a feeling of oppres-

sion and fullness in the thorax. Should distress occur during or after meals, walking about for a few moments usually gives relief. Patients with diaphragmatic hernia should be warned of the possibility of an obstruction and the necessity of surgical intervention.

If the symptoms are severe and the medical treatments give no relief, and if the hernia is of a type amenable to operation, surgery may be resorted to. The dangers of complications and of operation as well as the severity of the symptoms must all be considered. But surgery is not a panacea for hiatal hernias. Hedblom[22] points out that "Suture of the hiatus hernial ring may be relatively difficult on account of its relative inaccessibility, especially in deep-chested individuals and those with long, narrow chests. There is also a good chance of recurrence. The indications for operation on a hiatus hernia are therefore more exacting than any other type as such."

Thoracotomy and laparotomy or a combination of the two have been recommended. However, the abdominal approach is the one of choice for hiatal hernias. The hernia is completely reduced and the enlarged esophageal orifice of the diaphragm sutured to its normal size. Phrenicotomy has been used as a preliminary step to reduce the movements of the diaphragm and thus remove one of the complicating factors in operative procedures.

Harrington[41] has used phrenicotomy as a palliative treatment of cases of hernias in which radical surgical procedures are contraindicated because of the condition of the patient. In order to evaluate the amount of relief to be expected from phrenicotomy he recommends emphraxis by crushing or injecting the nerve before completely destroying it. Phrenicotomy may be of some benefit in the treatment of short esophageal hernias. The relaxation of one-half of the diaphragm may be sufficient to reduce pressure on the herniated portion of the stomach thus relieving the symptoms. However, this is doubtful since most of the symptoms are due to incompetency of the cardia. Fortunately most of the cases of hiatal hernias have only mild symptoms and surgical intervention is rarely indicated.

## CONCLUSIONS

1. Herniation of a portion of the fundus of the stomach through the esophageal hiatus is a more common condition than formerly thought. The increased number of roentgenologic examinations of the gastro-intestinal tract has resulted in more frequent diagnosis. Although no exact figures are obtainable in the literature, it is estimated that hiatal hernias represent approximately two-thirds of the non-traumatic and one-third of all the cases of diaphragmatic hernia.

2. Hiatal hernias may be divided anatomically into three groups:

1. Short esophageal hernias.
2. Para-esophageal hernias.
3. Hernias in which the esophagus is not shortened but the distal end is included in the hernial sac.

3. Short esophageal hernias may be the result of an abnormally far anterior position of the anlage of the stomach on the digestive tube, or to an idiopathic delay in the elongation of the esophagus. The other types may be congenital or acquired. Congenital cases result from a delay in the closure of the omental bursa. Acquired forms result from indirect trauma due to increased intra-abdominal pressure.

4. There is no characteristic symptom or syndrome diagnostic of this condition. The most common complaint is a dull pain under the ensiform, accompanied by regurgitation when in the recumbent position.

5. Physical signs are unsatisfactory.

6. Definite diagnosis may be made only with the roentgen ray. Careful fluoroscopic examinations are essential with the patient in the prone, supine and oblique positions. Menge's position is the most satisfactory. Roentgenograms are difficult to make.

7. There is a definite sequence in the types of stomachs as seen in the roentgenologic studies. This sequence goes from the normal stomach through the "infantile type" with the esophagus entering at the peak of the fundus, to the short esophageal hernias, and finally to the condition of thoracic stomach.

8. Roentgenologic differentiation of the three types of hiatal hernias is possible in typical cases.

9. Treatment consists in dietary and preventive management. Surgical repair may be resorted to in cases with severe, unrelieved symptoms. Reduction of short esophageal hernias is usually unsuccessful. Phrenicotomy may be done as a surgical measure in hiatal hernias where radical operation is contraindicated and as a pre-operative procedure for reduction. It may be of benefit in short esophageal hernias. Recurrences of hiatal hernias are frequent.

## REFERENCES

1 Paré, A.: Oeuvres. Paris, G. Buon. 1579.
2 Petit, J. L.: Malaise Des Os Oeuvres Complete. 1844.
3 Morgagni: Morgagni's Works. Volume III. Seats and Causes of Disease. (Cited by Bowditch, J. M. W. Morison and others.)
4 Bowditch, H. I.: A treatise on diaphragmatic hernia. Buffalo M. & S. J. 1: 65, 1853.
5 Lacher, L.: Ueber Zwerchfellshernien. Deutsches Arch. f. klin. Med. 27: 268, 1880.
6 Eppinger, H.: Pathologie des Zwerchfels. Supplement to Nothnagel's zu Spezielle Pathologie und Therapie. 1911. (Quoted by Giles.)
7 Giffin, H. Z.: The diagnosis of diaphragmatic hernia. Ann. Surg. 55: 388, 1912.
8 Breitner, B.: Zwerchfellshernien. Arch. f. klin. Chir. 117: 164, 1921. (Cited by Healy.)
9 Richards, L. G.: Nontraumatic hernia of the diaphragm. An embryologic viewpoint. Ann. Otol. Rhinol. & Laryng. 32: 1145 (Dec.) 1923.
10 Carman, and Fineman: Roentgenologic diagnosis of diaphragmatic hernia, with a report of seventeen cases. Radiology. 3: 26 (July) 1924.
11 Morrison, L. B.: Paper read before the New England Roentgen Ray Society, April 7, 1922.

12 Morrison, L. B.: Diaphragmatic hernia of fundus of stomach through the esophageal hiatus. J. A. M. A. 84: 161 (Jan. 17) 1925.
13 Healy, T. R.: Symptoms observed in fifty-three cases of non-traumatic diaphragmatic hernia. Am. J. Roentgenol. 13: 266 (March) 1925.
14 Morrison, L. B.: Personal Communication.
15 Pancoast, H. K., and Boles, R. S.: Nontraumatic left diaphragmatic hernia. Arch. Int. Med 38: 633, 1926.
16 Hedblom, C. A.: Diaphragmatic hernia. J. A. M. A. 85: 947 (Sept. 26) 1925.
17 Akerlund, A.: Diaphragmatic hernia of the esophageal hiatus. Acta Radiologica. 6: 1, 1926.
18 Truesdale, P. E.: Hernia of the diaphragm. Internat. Clin. 1: 14 (March) 1930.
Ibid.: Hernia in the diaphragm in children. J. A. M. A. 93: 1538 (Nov. 16) 1929.
Ibid.: Traumatic rupture as a sequence to congenital hernia of the diaphragm. Ann. Surg. 90: 654 (Oct.) 1929.
Ibid.: Diaphragmatic hernia in a child aged one year. J. A. M. A. 96: 847 (March 14) 1931.
Ibid.: Diaphragmatic hernia in an adult. New Eng. J. Med. 204: 1362 (June 25) 1931.
Ibid.: Symptoms and physical signs indicating hernia of the diaphragm with report of twelve cases treated by operation. Ann. Surg. 94: 528 (Aug.) 1931.
19 Greenwald, H. M., and Steiner, M.: Diaphragmatic hernia in infancy and childhood. Am. J. Dis. Child. 38: 361 (Aug.) 1929.
20 Ude, W. H., and Rigler, L. G.: Hernia of the diaphragm through the esophageal hiatus with report of nineteen cases. Minn. Med. 12: 751 (Dec.) 1929.
21 Ritvo, M.: Hernia of the stomach through the esophageal orifice of the diaphragm. J. A. M. A. 94: 15 (Jan.) 1930.
22 Hedblom, C. A.: Diaphragmatic hernia. Practice of Surgery, Lewis, 1931.
23 Harrington, S. W.: Diaphragmatic hernia. Arch. Surg. 16: 386, 1928.
24 Arey, L. B.: Developmental Anatomy. Philadelphia. W. B. Saunders Company. 1930.
25 Keith, Arthur: Human Embryology and Morphology. London, Arnold. 1923.
26 Bailey, P. A.: A case of thoracic stomach. Anat. Record. 17: 107, 1919.
27 Bright: Misplacement of the stomach. Guy's Hospital Report. 1: 598, 1836.
28 Bund, Rudolph: Ein Fall von rechtsseitiger Hernia diaphragmatica mit Austritt des Magens in den persistierenden Rezessus pneumato-entericus dexter. Frankfurt. Ztschr. f. Path. 21: 243, 1918. (Cited by C. C. Anderson, Healy.)
29 Tonndorf, W.: Diaphragmatic hernia as consequence of malformation of the esophagus. Deutsche Ztschr. f. Chir. 179: 259, 1923. (Quoted by L. Findlay and A. B. Kelley.)
30 Findlay, L., and Kelley, A. B.: Congenital shortening of the esophagus and the thoracic stomach resulting therefrom. J. Laryngol. & Otol. 46: 797 (Dec.) 1931.
31 Morison, J. M. W.: Diaphragmatic hernia. Proc. Roy. Soc. Med. Sect. Electro. Therap. 23: 23 (Sept.) 1930.
32 LeWald, L. T.: Roentgenological diagnosis of diaphragmatic hernia. Am. J. Roentgenol. 20: 423 (Nov.) 1928.
33 Jackson, Chevalier: Diaphragmatic pinchcock in so-called "Cardiospasm." Laryngoscope. 32: 139 (Feb.) 1922.
34 Mosher, H. P.: The lower end of the esophagus at birth and in the adult. J. Laryng. & Otol. 45: 161 (March) 1930.
35 LeWald, L. T.: Congenital absence of left half of the diaphragm. Arch. Surg. 14²: 332 (Jan.) 1927.
36 Kirklin, J. N.: Progress in roentgenologic diagnosis of diaphragmatic hernia. Proc. Staff Meet. Mayo Clin. 5: 197, 1930.
37 Cabot, R. C.: Cabot Case; Diaphragmatic hernia. New Eng. J. Med. 200: 88 (Jan. 10) 1929.
38 Crohn, B. B.: Posttraumatic diaphragmatic hernia. M. Clin. North America. 12: 583, 1928.
39 Giles, R. G.: Hernia of the diaphragm with report of a case. Texas State J. Med. 24: 418, 1928.
40 Schisler, E. J.: Diagnosis of congenital hernia with report of a case. J. Missouri M. A. 26: 228, 1929.
41 Harrington, S. W.: Phrenicotomy in the treatment of diaphragmatic hernia and tumors of the wall of the chest. Arch. Surg. 18: 561, 1929.

### Additional References

In addition to the above articles specifically referred to, the following papers were reviewed.

Abell, I.: False hernia of diaphragm with dilatation of cardia and esophagus. S. Clin. North America. 10: 909 (Aug.) 1930.
Adams, A. W.: Thoracic stomachs. Study of pharyngeal pouch and cardiospasm, with report of three cases. Brit. M. J. 2: 208 (Aug. 9) 1930.
Adams, A. W.: Hernia of stomach into right pleural cavity, pulmonary abscess in right lower lobe. J. Laryng. & Otol. 46: 693 (Oct.) 1931. (Reports case with a short esophagus.)
Ager, L. C.: Diaphragmatic hernia. U. S. Vet. Bur. M. Bull. 6: 971 (Nov.) 1930.
Anderson, C. C.: Hernia through esophageal orifice of diaphragm. Radiology. 14: 602 (June) 1930.
Angwin, W. A.: Diaphragmatic hernia. U. S. Nav. M. Bull. 28: 106 (Jan.) 1930.
Bettman, R. B., and Hess, J. H.: Diaphragmatic hernia in infant. Ann. Surg. 93: 1274 (June) 1931.
Bevan, A. D.: Diaphragmatic hernia. Arch. Surg. 1: 23 (July) 1920.
Brown, H. P.: Diaphragmatic hernia. S. Clin. North America 9: 1191 (Oct.) 1929.
Carrington, G. L.: Diaphragmatic hernia repair by two stage operation employing preliminary extra-pleural thoracoplasty. Ann. Surg. 89: 512, 1929.
Chapman, E. F.: Congenital diaphragmatic hernia. Brit. M. J. 2: 500 (Sept. 14) 1929.
Dansey, J. W.: Congenital diaphragmatic hernia. M. J. Australia. 2: 920 (Dec. 28) 1929.
Dealy, F. N.: Right-sided traumatic hernia. Am. J. Surg. 8: 1033 (May) 1930.
Dodds, G. H., and Pocock, J. A.: Right-sided true diaphragmatic hernia with unusual features. Arch. Dis. Childhood. 5: 233 (June) 1930.
Donovan, E. J.: Repairs in infant of four months of diaphragmatic hernia. S. Clin. North America. 11: 521 (June) 1931.
Elward, J. F., and Otell, L. S.: Nontraumatic diaphragmatic hernia. Am. J. Roentgenol. 22: 535, 1929.
Fineman, S., and Connor, H. M.: Right diaphragmatic hernia of the short esophagus type. Am. J. Med. Sc. 167: 672 (May) 1924.
Fortier, L. A., and Gately, T. T.: Obstruction of esophagus caused by diaphragmatic hernia. Texas State J. Med. 27: 488 (Nov.) 1931.
Friedenwald, J., and Fieldman, M.: Report on interesting type of diaphragmatic hernia of the cardia of the stomach through the esophageal orifice. Tr. Asso. Am. Phys. 11: 321, 1925. Also Am. J. Med. Sc. 170: 263, 1925.
Geyman, M. J.: Right diaphragmatic hernia. Radiology. 16: 483 (April) 1931.
Gibson, F. S.: Diagnosis of hernia (traumatic) with acute destruction. J. A. M. A. 93: 1719, 1929.
Harrington, S. W.: Diaphragmatic hernia associated with traumatic gastric erosion and ulcer. Surg. Gynec. Abst. 51: 504 (Oct.) 1930.
Hipsley, P. L.: Congenital diaphragmatic hernia in young girl. M. J. Australia. 1: 200 (Feb.) 1931.
Hoover, W. B.: Dysphagia. Clinical aspects with cases illustrating some pathologic conditions of the esophagus. Surg. Clin. North America. 12: 713 (June) 1932.
Hume, J. B.: Congenital diaphragmatic hernia. Brit. J. Surg. 10: 207, 1922. Reports three cases, cites 35 from Eng. & Am. Lit. 1910-1920.
Hume, J. B.: Diaphragmatic hernia. Brit. J. Surg. 19: 527 (April) 1932.
Jenkinson, E. L.: Absence of half of diaphragm. Thoracic stomach; diaphragmatic hernia. Am. J. Roentgenol. 26: 899 (Dec.) 1931.
Jenkinson, E. L.: Lesions of the diaphragm. Am. J. Roentgenol. 14: 16 (July) 1925.
Johnson, H., and Bower, A. G.: Strangulated hernia in infant. Case. California & West. Med. 36: 48 (Jan.) 1932.
Janes, R. M.: Repair of diaphragmatic hernia with fascia lata Canad. M. A. J. 24:421 (March) 1931.
Jordan, S. M.: Importance of functional tranquillity in abnormal mechanism of digestive apparatus. S. Clin. North America. 12: 707 (June) 1932.
Keller, W. L.: Massive diaphragmatic hernia without diagnostic symptoms. Am. J. Surg. 8: 598 (March) 1930.
Kelly, A. B.: Congenital stenosis in children associated with diaphragmatic hernia of stomach. Proc. Roy. Soc. Med. (Sect. Laryng.). 23: 71 (Sept.) 1930. Also J. Laryng. & Otol. 45:680 (Oct.) 1930.
Knight, G. C.: Diaphragmatic hernia with report of case. St. Barth. Hosp. J. 39: 135 (April) 1932.
Lafield, W. A.: Hernia through esophageal opening of diaphragm. Case of right-sided hernia. Yale J. Biol. & Med. 3: 165 (Dec.) 1930.
Lemon, W. S.: The function of the diaphragm. Arch. Surg. 17: 840 (Nov.) 1928.
LeWald, L. T.: Thoracic stomach: Differentiation from eventration and hernia of diaphragm. Radiology. 3: 91 (Aug.) 1924. Reports two cases of thoracic stomach and points out that stomach never in thorax and diaphragm has developed normally.
Lightwood, R.: Right-sided diaphragmatic hernia. Proc. Roy. Soc. Med. 25: 783 (April) 1932.
Lilienthal, H.: Diaphragmatic hernia of liver. S. Clin. North America. 11: 475 (June) 1931.
Lord, F. T.: Case of diaphragmatic hernia with remarks on diagnosis. M. Clin. North America. 13: 67 (July) 1929.
Lyons, C. G.: Thoracic stomach, Case. U. S. Vet. Bur. M. Bull. 6: 65, 1930. Am. J. Roentgenology. 23: 67 (Jan.) 1930.
Martin, C. P.: So-called thoracic stomach, two cases. Irish J. M. Sc. 6th Series. P. 564 (Oct.) 1931.
Moore, A. B., and Kirklin, B. R.: Progress in roentgenologic diagnosis of diaphragmatic hernia. J. A. M. A. 95: 1966 (Dec. 27) 1930.
Morris, H.: Thoracic stomach. Radiology. 13: 265 (Sept.) 1929.
Overholt, R. H.: Phrenic nerve stimulation under fluoroscope as aid in diagnosis. Ann. Surg. 91: 381 (March) 1930.
Owen, C. C.: Two unusual cases of eventration of the diaphragm in routine roentgenography. Radiology. 18: 361 (Feb.) 1932.

Richardson, E. P.: Hernia through esophageal orifice. Tr. South S. A. 41: 14, 1928. Also Surg. Gynec. & Obst. 49: 129 (Aug.) 1929.
Roberts, R. E.: A case of thoracic stomach. Brit. J. Radiol. 32: 17, 1927.
Salomon, A. V.: Diaphragmatic hernia accompanied by dextrocardia, Case. Arch. Pediat. 48: 802 (Dec.) 1931.
Sellars, T. H.: Thoracic exposure of diaphragm and lower esophagus. Proc. Roy. Soc. Med. 25: 1004 (May) 1932.
Shephard, J. H.: Cardiospasm. (All cardiospasm, no hernia.) Calif. & West. Med. 35: 422 (Dec.) 1931.
Smith, A. D.: Congenital diaphragmatic hernia. M. J. Australia. 2: 333 (Sept. 6) 1930.
Soresi, A. L.: Diaphragmatic hernia. Ann. Surg. 69: 254, 1919.
Sussman, M. L.: Congenital atresia of esophagus. Case. Am. J. Roentgenol. 26: 894 (Dec.) 1931.
Traut, E. F.: Hernia of diaphragm. M. Clin. North America. 14: 1123, 1930-31.
Truesdale, P. E.: Traumatic rupture as sequence to congenital hernia of diaphragm, with experimental study of its mechanism and effects of phrenicotomy. Ann. Surg. 90: 654 (Oct.) 1929.
Truesdale, P. E.: Hernia of the diaphragm. Internat. Clin. 1: 14 (March) 1930.
Whedon, E.: Symptoms of diaphragmatic hernia of stomach. Colorado Med. 27: 108 (March) 1930.
Woolsey, J. H.: Diaphragmatic hernia. J. A. M. A. 89: 2245, 1927.

# PRACTICAL FEATURES IN THE STUDY AND TREATMENT OF ANXIETY STATES*

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ONE of the most interesting and common symptom-pictures of nerves is that which is comprised in attacks of dyspnea, palpitation, sweating, vertigo, precordial discomfort and often gastro-intestinal manifestations with the predominating emotional accompaniment of apprehensiveness—a vague fear of something going to happen. Its force is best described in the term *angst*—or anxiety. The first person to describe this symptom-picture was a German by the name of Hecker[1] in 1893. He classified it among the symptoms of neurasthenia. Little attention was paid to the observation till 1909 when Freud described the same condition and labeled it anxiety neurosis. In 1912 and 1920 he elaborated[2] upon the theme. As you know, Freud attempted to divide what had hitherto been known as neurasthenia and hysteria into physioneuroses and psychoneuroses. Under the physioneuroses he put neurasthenia (irritable weakness) and anxiety neurosis. He attributed these nervous conditions to some interference with the ordinary physiology of sex functioning. Neurasthenia he ascribed to masturbation, or autoerotism, and anxiety neuroses to frustraneous sexual excitement brought about by a great variety of unfulfilled stimulations and erotic activities. Upon this principle he built an elaborate and ingenious theory which certainly sounds plausible to those who have never spent any thought upon the mechanism of anxiety states, or the common variety of situations and facts that may give rise to the same symptom complex. A certain number of our profession following Freud's lead have approached these patients unconsciously determined to find some malfunctioning in the sex life to account for the symptomatology. For example, the practice of coitus interruptus, which is one of the commonest birth control methods, has been condemned wholesale on the theory that it is productive of anxiety attacks. Again, anxiety attacks in male and female have been treated with prescriptions of wholesale sex activities on the supposition that the patient was suffering from repressed libido with disastrous results to the health and happiness, and full-fledged psychosis as sequelae in many instances. In other words, Freud's theory has been made a Procrustes bed to which patients suffering from anxiety attacks must be made to fit. While it is undoubtedly true that frustraneous sexual excitement may give rise to states of anxiety, it is not by any means the one and only cause of such conditions as Freud led us to believe for many years. It is interesting to note that in Freud's "Inhibition, Symptom and Anxiety" published in 1927[3], he has changed his concept of anxiety neuroses which he now believes involve more than just a repressed and unsatisfied libido.

Meantime the medical profession having become aware of this symptom-picture of anxiety states and unwilling to accept or unaware of Freud's conceptions groped its way toward other therapy. Called to see such a patient in the midst of an attack the physician sees an individual sitting up in bed gasping for breath, with wide staring eyes, rapid pulse, sweat on the brow and hears the exclamation, "Doctor, I'm dying. Do something." An equally terrified family stand about opening windows, fanning, rushing for hot water bag or ice-cap. Their terror is grafted upon that of the patient. The physician ushered into this scene listens to a rapid, disturbed heart with an extrasystole or a sinus arrhythmia and speaks as follows: "I think this is nothing serious. You have a nervous heart that is skipping beats. I wouldn't say there is any grave heart disease, but you'd better be careful. Go slowly. Don't run for a street car. Lie down if possible for a while after each meal." He may give digitalis and put the patient on a restricted cardiac régime. In this way a third scare is grafted upon the two which the patient has already had. One

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might just as well tell a nervous patient that all his heart valves are leaking like spigots as to damn with such faint praise this organ so fundamental to our well-being.

A second method of therapeutic approach is to tell these patients that gas in the stomach is pressing the diaphragm upward, and put them on diets and tincture of belladonna. The result is that the patient's attention becomes fixed on gas, and he gets as panicky with every rumbling of his viscera as a nervous woman in midsummer when a cloud passes over the face of the sun.

A third therapeutic accusation is directed toward that much abused and misunderstood organ, the thyroid gland. Before we had the basal metabolic test, the anxiety neurotic was treated most indiscriminately as a case of hyperthyroidism. Today we are a little more conservative in our operative procedures of ligation and extirpation. It is interesting to note that anxiety attacks show a marked increase in mass health during periods of mass excitement and worry for any reason. For example, in 1918 and 1919, during the "flu" epidemic, these cases were very, very common in private and dispensary practice. We called them "post flu hearts" in those days. But we may well ask ourselves how much of this symptomatology was due to actual influenza heart damage, and how much of it was associated with a high pitch of emotional excitement due to an epidemic that rivaled the black plague in its mortality rate. During the year 1926 when America was sitting comfortably on an inflated economic foundation, we registered only 80 of these patients in the Dispensary of the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital. In 1932 when we began to feel the pressure of deflation we registered 232 cases of pure anxiety states in our Henry Phipps Psychiatric Dispensary. We shall go over that figure for 1933.

It would, therefore, seem reasonable to study the symptom-complex of anxiety attacks to see what lies behind in the rank and file of cases. The complaints of palpitation, fluttering, giddiness, heart turning over, sinking spells, shaking, sweating, and flushing, weakness, constriction in the chest, pain around the heart, substernal pressure, shortness of breath, tightness in the throat, fear of being alone, etc., are symptoms with which we all are familiar in states of fear. The receipt of bad news, the necessity of speaking in public, the finding of ourselves in situations of danger call forth in each of us these physiological manifestations of fear. Cannon of Harvard has described for us in detail what fear does to animal physiology. We pay no attention to these distressing cardiovascular and respiratory and gastro-intestinal body protests because they seem to be a reasonable accompaniment to a definite emotional state. We are afraid of something concrete and perhaps objective such as death of a loved one, financial disaster, personal disgrace, physical danger. Similar body protests in the anxiety patient seem unreasonable because we see no concrete thing happening to him, and this sounds unreasonable to us. But suppose we go back and study the personality of this individual, and see if we can find a reason why he behaves in this way. In doing so we discover that he is invariably a person who all through life has been timid, supersensitive, overanxious, an "easy-worrier", extremely conscientious, serious-minded. One never finds anxiety states in a phlegmatic temperament. Such an anxiety temperament, prone to go out to meet trouble instead of waiting until it gets to him, is bound to react in an exaggerated way to every sling and arrow of outrageous fortune which he encounters in the pathway of life. He takes life very seriously and very hard. Circumstances which another temperament would make light of and pass over, this kind of an individual broods over, takes to heart, and worries about. His family may not realize it. His friends and business associates may not know it, but the process of temperamental fermentation goes on nevertheless. For weeks and months preceding the condition that we call an anxiety attack, the soil of his conscious processes has been ploughed and harrowed for the reception of some shock or sudden experience. The shock or experience may seem trivial and insignificant to us as we look at it in the light of a single incident or episode. But to him it is the culmination of a long period of anxious worry and mental unrest. This precipitating factor may be an operation, the illness of a child, a financial loss, a demotion, a promotion to a position of responsibility, an accident. One sees the condition frequently in children following a tonsil and adenoid operation which has been preceded for weeks by family conversations about death from taking ether, and hemorrhages, and the terrible things that happen to people in hospitals. Such talk affects an impressionable child, but rolls off a phlegmatic one. Imagine what happens to such a man, woman or child in an anxious state of mind when the physician darkly threatens the patient with fears of some grave systemic condition, and begins a long and tedious series of examinations and hospital experiences and treatments. The following, for example, are some of the diagnostic suspicions entertained by our profession concerning the 232 anxiety neurotics seen in our Dispensary during the year 1932.

| A. Where cardiac symptoms were prominent. | B. Where G. I. symptoms were prominent. |
|---|---|
| 1. Angina pectoris. | 1. Gastro-intestinal disease. |
| 2. Pseudo-angina pectoris. | 2. Fallen stomach (dropped). |
| 3. Heart trouble. | 3. Spastic colon, g. i. t. stomach. |
| 4. Rapid heart. | 4. Chronic constipation. |
| 5. Heart murmur. | 5. Gastric ulcers. |
| 6. Nervous heart. | 6. Gall bladder disease. |
| 7. "Irritable" or "soldier's" heart. | 7. Appendicitis. |
| 8. High blood pressure. | 8. Gastric neurosis. |
| 9. Low blood pressure. | 9. Cecal stasis. |
| 10. Fast pulse. | 10. Gastropathy. |
| 11. Essential hypertension. | 11. Functional dyspepsia. |
| 12. Effort syndrome. | 12. Nervous indigestion. |
| 13. Vasomotor instability. | |

Needless to say all these conditions were ruled out by appropriate examinations in various departments of our General Dispensary.

The following are some of the precipitating factors in the anxiety outbursts of these 232 cases:

Removal of the teeth; operations, appendectomy.

Deaths in family, especially when due to heart trouble.

Accidental shooting.

Attempted murderous assault.

Automobile accident.

Fall from wagon.

Influenza.

Began during the War.

Criminal abortions (2) following pregnancy.

Quarrel with neighbors.

Knowledge of husband's infidelity.

Knowledge that husband had syphilis.

Worry over step-daughter's sex irregularities.

Broken engagement.

Irregular or no work. Increasing number of family beyond level of economic maintenance. Strivings beyond capacity, work under pressure, night courses, and change of vocational activity.

Sex conflicts and marital difficulties, infidelity, in two cases frigidity of wife; dislike of alcoholic husband; broken engagement; illicit sex relations; conflict about birth control, fear of pregnancy. In only two cases did frustraneous sex excitement occupy a prominent place. The use of contraceptives was admitted by many. The sex difficulties seemed to be more important when there were sex irregularities with conflict, or when "in-laws" were interfering.

Alcoholism in husband or parent; mother deserted family; insurance and industrial compensation factors; religious conflicts of Catholic-Protestant marriages; jealousy of younger sibling; father in trouble about bootlegging; husband unstable and depressed.

And here are some facts concerning the personality and intellectual assets and biological instabilities of this same group of patients:

Over two-thirds of the patients gave definite histories of neurotic tendencies or of neuropathic constitutions dating back to childhood; e.g., bed-wetting, nail-biting; "scariness," timidity, jumpiness, night terrors, fear of storms; fear of illness, rumination in infancy, fussiness over food, easy apprehensiveness and excitability during the school period; hives, eczema, hay fever, asthma.

About three-fourths of the patients were mentally retarded or of borderline (twelve to fourteen) intelligence. There were few colored patients.

From the standpoint of treatment these 232 cases may be divided into three different methods of medical approach, dependent upon whether the physician considered their condition due to cardiac, gastro-intestinal, thyroid or menopausal etiology.

Cardiac approaches:

Rest from two weeks to months.
Digitalis (one case for three years).
Sedatives — luminal, bromide, and codeine, morphia, spirits of ammonia, whisky.
No exercise—or light exercise.

Gastro-intestinal approaches:

X-ray series.
Drugs: Belladonna or hydrochloric acid.
Diets: Sippy diet for gastric ulcer.
Treated for stomach trouble 30 years.
Tonics—fresh air.
Enemata for indigestion.

Thyroid approaches:

Thyroidectomy, iodine, rest cures.

Miscellaneous approaches:

Ovarian extract, dilatation and curettage; endocrine therapy; teeth extractions; protein sensitization.

The first point in the treatment of a patient in an anxiety state is to settle the issue of heart or stomach or thyroid condition by a thorough examination of the physiological functioning of which the patient complains. This examination should be completed as speedily as possible. To drag out such investigations for weeks and months trying digitalis and belladonna and thyroid and ovarian extracts is a psychologically fatal procedure. The patient feels no better and suspects all the way along that the doctor is not sure of his ground and is feeling his way along in insecurity. The result is that he goes to another doctor, and still to another until his money is exhausted and he is unable to carry on his work, and finally arrives in a dispensary. This is the story of these 232 patients already mentioned. If the physician is not sure of a heart, or gastro-intestinal condition, excellent specialists are available in

diagnostic clinics and dispensaries with equipment for electrocardiogram and chemical analysis. The diagnostic clinic and the dispensary do not wish to treat these patients. They are glad to study and attempt a diagnosis, and send these patients back to the practitioner to treat.

Having ruled out any so-called lesional pathology the next step is to convince the patient that these body protests of heart or gastro-intestinal tract represent emotional states, the nature and content of which it is the doctor's business to discover and interpret for his patient. One must remember that all of us are very suggestible as human beings, but the anxiety neurotic is especially suggestible. It does no good to tell such a palpitating individual that his heart is sound, and then give him a tonic or some other bottle of medicine, and tell him to go slowly, to rest as much as possible, or to take a trip somewhere. I have had these patients say again and again, "Yes, the doctor told me my heart was all right, but he gave me medicine and told me to rest, and I thought he was keeping something from me." The best educational process is sending such a patient right straight back to work, or to send him into crowds, or whatever else he is afraid of, telling him that he is quite likely to have other seizures, that he will feel wretchedly apprehensive, but if he will stick it out he will cure his fear, and he alone can do the trick. Years ago when we had horses and steam rollers the same sort of educational process worked in getting the horse over being afraid of this terrifying object in the road. One drove him close to it, let him shake and tremble and jump, and eventually he became so used to the engine that he did not even shy. Change of scene with its diversions merely postpones the day when the patient must return and face the situation which gave rise to his fears. Moreover, one must remember that he takes his personality and biology with him to Atlantic City or Europe.

The next step is to discover the factors of situation or domestic relations or vocational misfitting that have upset the patient's equilibrium. The physician who reads psychoanalytic literature is apt to go about this by looking for some very obscure and complex mental conflict. As a matter of fact the common factors lying behind anxiety states are relatively simple. It is almost useless to ask such a patient if anything is worrying him, for he usually gives a perfectly smooth story in the negative, not because he is secretive, but because he is honestly unaware of what is upsetting him. He may be aware of situations that are uncomfortable, but quite unaware that they are having any affect on him. It is up to us to take a little time to go into such commonplace matters as fear of losing a job, iliness in the family, sudden deaths from stroke and heart disease, the losing of a home through inability to meet building loan payments, an accumulation of small debts, crowded living conditions associated with having to bring indigent relatives into the family home. Having discovered these factors it is not enough to tell such a patient not to worry, and to exercise self-control. The facts revealed may make him feel ashamed that such matters could get a rise out of him. Right here it is very important to put him at ease with regard to the frequency with which similar irritations upset all of us. It is helpful to take a little time to explain to him that under situations of discomfort even you and I, his physicians, are conscious of our bodies protesting in palpitation, sweating, shortness of breath, nausea. This makes him accept his symptoms as natural biological processes that others have, and desensitizes him to scare, in a quiet, matter-of-fact manner.

Having given the patient dramatic doses of reassurance with plausible explanations, the next step is to get hold of husband or wife or parents and explain matters to them. It is well to do this in the patient's presence so that he does not think that you are telling one thing to him and another to his family. Elicit the family's intelligent coöperation in treating the next anxiety attack in as casual a manner as possible. Prescribe emphatically that the next time the patient has an attack in movie, at home, or on the street car the family is to sit quietly, not yielding to the patient's demands for spirits of ammonia, opening of windows, fanning, rubbing of hands and feet, and all the many procedures, environment goes through in its own fear of the patient's apprehensiveness, that he or she is dying. Such activities merely graft another scare upon the one which the patient already has. If relatives are given these instructions in the patient's presence they have a chance to carry them out backed up by the doctor's recommendations, and the patient cannot accuse them of being cold and heartless. All these details may sound childish and a waste of time, but they are frequently necessary to produce the psychological reassurance that the patient needs to calm his panic. If the patient really wants to be well and is not using his attacks as a cudgel over the heads of the environment, and if the family will carry out their share of the treatment, the condition subsides with surprising rapidity. Of course, there are a certain number of patients who do not want to be well, and there are a certain number of patients in whom anxiety attacks are not psychoneurotic manifestations but episodes that occur as part of, or prodromal features, of a major psychosis, such as the manic-depressive and the schizophrenic, or dementia praecox group, or as part of a psychopathic personality picture. Of the 232 cases mentioned above, 65 per cent responded in a matter of weeks or a month or two to the treatment procedures I have outlined. The

remaining 35 per cent fell into the groupings of unmodifiable human material for reasons just described. Let me outline a case or two as typical of symptom-picture and treatment.

Mrs. P. is a woman of 28, referred from our Medical Dispensary, April 1932, for palpitations, constant indigestion, belching, afraid to remain in the house alone, fear of crowds, thinks the house is going to cave in on her, afraid of dying when she has these spells.

Her present symptoms began six months ago when her husband lost a position which he had had for twelve years, and she and her husband were obliged to reduce the standards of living to which they had been accustomed.

Fluoroscopic examination showed spastic colon. She had been taking tincture of belladonna and alkaline powders. In March she was seen in the Protein Clinic for patches of eczema.

In the background we have a "scary, canoe-like" personality, who as a little child had occasional outbursts of eczema under the strain of changing schools or speed tests. She has, also, had one or two outbursts of asthma whenever passing through a period of worry occasioned by her erratic and alcoholic father.

Treatment: The condition was explained to the patient and family with resulting elimination of symptoms in three weeks, and no subsequent return of her former invalidism.

Earl H., aged 32, a worker of the Bethlehem Steel Company was referred to our Psychiatric Dispensary, because one morning, several weeks before, he ran into the Accident Room of our Hospital in an attack of belching, palpitation, dyspnea and apprehensiveness.

The present illness began in September, 1932. From that time until he came into our Accident Room he was under almost continuous treatment from a gastro-intestinal standpoint. All examinations were negative. He was tried on belladonna and diets and bromide and other nerve medicine; every time this therapy failed he became even more panicky and upset than before. Within two weeks time his sick benefit would have expired.

In the background of the condition we find a man who had worked regularly for the Bethlehem Steel Company since twenty years of age. He is a skilled workman. Last June, by reason of two cuts he was unable to keep up payment on a home which he had been trying to buy during the last three years, and to which he and his family were much attached. All summer they looked for quarters, and in the fall found a broken-down shack of a place which they were able to get for very little rent because the owner could do nothing else with it. A friend of the family who had always paid board until this time lost his job in September, 1932, and Mr. H. felt that he should keep him. The patient did not complain of any of these circumstances that I have mentioned. His complaint was of his stomach and the palpitations. When asked in general about worries and cares he denied the same in a cheerful way saying, "We all have our troubles and mine are no worse than anybody else's, I guess. If I were feeling well, I would not mind anything." This man was put directly back on his job, being told that he would feel badly, that he would have more attacks, but if he would see it through he would not regret the effort it took to break up these nervous habits. He returned to work five days after his condition was explained to him and has not lost a day since then.

In summary then, anxiety attacks, or anxiety neuroses, are common and relatively benign nervous conditions that the practitioner can handle effectively if he is not too befogged by the complexities of psychoanalytic literature, and if he is willing to take a little time and interest in understanding the personality and situational background of his respective patients. The condition may, however, fall into chronic invalidism and a profound doctor habit status if treated along the mechanical lines of rest. drugs, and specific intensive therapy directed against physiological systems that happen to be registering protests against emotional strains of which the patient may or may not be aware.

REFERENCES

1 Hecker, E.: Ueber larvirte und abortive Angstszustände bei Neurasthenie. Centralbl. f. Nervenh. N. F. 4: 565, 1893.
2 Freud, Sigmund: Selected Papers on Hysteria and Other Psychoneuroses. Nervous and Mental Disease Monograph Series, No. 4. Nervous and Mental Disease Publishing Company, 1920.
3 Clark, L. Pierce: Inhibition, Symptom, and Anxiety. Arch. Psychoanal. 1: 461 (Jan.) 1927. Published by The Psychoanalytic Institute, Stamford, Connecticut.

# THE TREATMENT OF PEPTIC ULCER COMPLICATED BY HYPERSECRETION

BY EDWARD S. EMERY, JR., M.D.*

THE object of this paper is to point out that there is a certain group of patients with ulcer, who are resistant to all our present means of therapy and may, indeed, be made worse if the more radical forms of treatment are used. It is now pretty generally recognized that ulcer is a chronic disease and that none of our present methods of treatment are curative. Furthermore, the study of a large number of patients with ulcer shows that the greater proportion can be kept comfortable and that the results of treatment by different methods are much the same, provided the same type of case is treated[1]. Yet there has been a growing tendency to use more radical forms of surgery with the hope of curing a greater number of patients.

If the simpler forms of treatment do not prove satisfactory, it is natural to search for better results with more radical methods. However, radical treatment is contraindicated in certain patients of which the following cases are examples:

CASE I, A. F., medical No. 29971, an Italian laborer of 30 years, first entered the hospital in April, 1927. For two and a half years he had been having typical symptoms of ulcer.

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Physical examination was essentially negative, his weight at this time being 140. The x-rays showed a small, constantly deformed and flattened cap. There was no evidence of obstruction. A gastric analysis showed a free acidity of 45 and a total acidity of 65. Surgery was advised because it was thought that "the patient's occupation and general mental attitude made it inadvisable to try medical treatment." A Horsley pyloroplasty was done and, after a stormy convalescence, the patient was discharged from the hospital twenty days later.

Reëxamination by the x-ray showed a hypertonic, somewhat dilated stomach with a six-hour residue estimated at fifteen per cent. Gastric outline was smooth except at the pyloric canal which was irregular and distorted. The barium emptied poorly through the narrow channel.

He was observed in the Out-Patient Department of another hospital until August, 1927, when he re-entered the Surgical Service of the Peter Bent Brigham Hospital. Three weeks before re-entry he began to experience dull, persistent pain in the lower abdomen. A gastro-enterostomy was performed and an x-ray examination two weeks later showed the stoma to be functioning well.

However, the symptoms returned shortly after discharge, and an x-ray done at the Boston Dispensary in the summer of 1929 showed the presence of a jejunal ulcer. Because the pain became severe he was readmitted to the Surgical Service of the Peter Bent Brigham Hospital in September, 1929. An x-ray at this time showed a narrowing and irregularity of the jejunum and a crater about 1½ cm. distal to the stoma. Another operation was performed by which the gastro-enterostomy was undone and one-third of the stomach was resected. A gastric analysis previous to this operation showed a free acidity of 101 and a total acidity of 110.

As the patient continued to have pain and did not gain in weight, he was readmitted to the Medical Service in October, 1931. An x-ray at this time showed that the gastro-enterostomy was functioning intermittently. Palpation under the fluoroscope showed two craters apparently in the stomach along the resected end just distal to the stoma. A gastric analysis showed a free acidity of 116 and a total acidity of 126. He was placed on a careful Sippy régime and remained in the hospital for three months. During this time he gained some strength and put on ten pounds in weight. However, his symptoms returned shortly after discharge. He is now unable to work and is worse today than when he was first seen in 1927.

Case II, B. W., medical No. 31512, a Jewish upholsterer first entered the Medical Service in December, 1927 at the age of 28. He had been suffering for four years from epigastric distress after meals, relieved with food and soda. He had been given a dietary treatment for duodenal ulcer three years previously in the Michael Reese Hospital in Chicago. Physical examination at the time of entry was essentially negative. A gastric analysis showed a free acidity of 90 and a total acidity of 108. He was placed on a Sippy régime, and after two weeks I made the following note: "The patient now shows a definite retention which will probably disappear to some extent on further treatment. However, it seems up to the patient to decide whether he wants to take operation now, or see if the retention will decrease on further treatment."

On January 2, the surgeons reported as follows: "The patient appears to have had two fair trials of medical treatment with many intervening months of partial treatment without result and surgery should be offered as a possible means of relief." At the time of operation the scar of a large ulcer was found in the first portion of the duodenum, just distal to the pylorus. It could not be completely excised and the end of the duodenum was turned in and sutured. A posterior Polya operation was done, the free end of the stomach being pulled through the transverse mesocolon and joined to the jejunum by a lateral anastomosis.

The gastric symptoms recurred two weeks after discharge and he re-entered the Medical Service in August, 1928, because of a hemorrhage. He was discharged after a few weeks but continued to have pain and frequent hemorrhages. After four medical admissions for pain and bleeding, the patient was explored in April, 1929, and no cause for the bleeding was found. However, the symptoms continued in spite of all kinds of medical treatment, including mucin and x-ray therapy. The gastric acidity during these years ranged from 40 to 70. In June, 1932 an x-ray examination revealed the presence of a jejunal ulcer and he was readmitted to the Surgical Service in August, 1932.

At operation, many adhesions had to be removed and so much difficulty was encountered that the whole stomach could not be sufficiently mobilized to attempt a true sub-total gastrectomy. Consequently, three-fourths of the stomach was removed making a direct anastomosis to a long loop of the jejunum. He was discharged twenty-three days after the operation and, at this time, a moderate amount of free hydrochloric acid was found to be present. Since then the gastric acidity has increased to its preoperative value. The symptoms have continued and the latest x-ray examination shows a possible gastric ulcer.

Case III, P. J. M., surgical No. 17643, a machinist, first entered the Surgical Service in October, 1922 at the age of 32, for osteomyelitis. He also complained of epigastric distress coming two hours after meals and relieved with food and soda.

An x-ray showed a duodenal ulcer with hyperperistalsis. A gastric analysis revealed a free acidity of 80 and a total acidity of 90. The rest of the laboratory findings were negative. The surgeons performed a gastroenterostomy with a plication of the pylorus which gave complete relief for two years. Then a massive hemorrhage occurred for which he was admitted to the Medical Service in November, 1924. No gastric analysis was done at this time but he was put on a Sippy régime and aspirations showed a total acidity as high as 80.

Except for the chronic osteomyelitis of the leg, the patient remained well until December, 1928, at which time his gastric symptoms returned. An x-ray showed that the jejunum just distal to the stoma was narrow and irregular with a crater on the lateral aspect. He was started on a Sippy régime, shortly after which he developed diarrhea. This continued until he entered the Medical Service in October, 1929. X-rays revealed the presence of a gastrocolic fistula and he was transferred to the Surgical Service. Gastric analysis showed a free acidity of 60 and a total acidity of 92.

The old gastroenterostomy was demounted and the gastrocolic fistula closed. The patient was symptom-free for five months and then began to experience a sense of fullness in the epigastrium, loss of appetite and belching of gas. This was followed by vomiting. He re-entered the Surgical Service in May, 1930, where it was found that he had developed a marked pyloric obstruction for which a gastroenterostomy was again performed.

Following this operation he was symptom-free for seven months at which time he developed a painless diarrhea. This continued unchecked and in September, 1931, the x-ray revealed another gastrocolic fistula. Gastric analysis showed a free acidity of 60 and a total acidity of 90. On rest in bed the diar-

rhea practically ceased and the surgeons were, therefore, loath to operate. He was discharged and continued in much the same condition until February, 1933, when he showed so much loss of strength that it seemed wise to try further surgery. At this operation the opening into the colon was closed and a subtotal gastrectomy was performed, only enough stomach being left to permit doing an anastomosis with the intestine. As the fistula had caused a constriction of the jejunum with a resulting dilatation of the proximal loop, the jejunum was transected, the constriction removed and the distal cut end sutured to the stomach. The proximal end of the dilated portion was then closed and a lateral anastomosis made with the normal portion of the jejunum. The patient made a good recovery.

Eight days after the operation a gastric analysis showed a free acidity of 27 and a total acidity of 34. The patient was then in a convalescent home for six weeks and has done very well up to the present time.

Case IV. J. W. N., medical No. 23383, a steel worker, first entered the hospital in March, 1924, at the age of 42. The present illness began nine years previously with epigastric distress and vomiting two to three hours after eating. Physical examination was essentially negative. A gastric analysis showed 93 points of free hydrochloric acid and a total acidity of 110. The patient was anemic but the blood returned to normal during his ten days in the hospital. An x-ray examination showed that the cap was small, definitely defective resembling somewhat the appearance of a pine tree and there was a definite tender point over the cap. This was regarded as indicating ulcer.

He was given six meals of bland food and some alkaline powders and was discharged improved. He later returned to the Out-Patient Department for recurring epigastric discomfort and was readmitted to the wards in February, 1932. Physical examination was again negative. The x-rays were unchanged. A gastric analysis gave a free acidity of 105 and a total acidity of 125. He was started on a Sippy régime, on which he developed a marked alkalosis. As many of the staff felt that medical treatment had proved unsatisfactory, he was transferred to the Surgical Service and a transection through the proximal part of the antrum was done and a gastro-jejunostomy performed after the lower stump was inverted, according to the Billroth No. 2 procedure. The patient made an uneventful recovery and was discharged from the hospital on March 15, 1932, twenty-one days after the operation.

Shortly after discharge the patient developed mild gas pains, and a week later had typical symptoms of ulcer. The pain rapidly became more severe and he was readmitted to the Medical Service on April 2. An x-ray examination revealed that the barium emptied rapidly and well through the posterior gastroenterostomy but there was a large crater about 2 cm. distal to the stoma. Some of the barium filled the proximal loop, which appeared dilated, and there was a small six-hour residue in the region of the duodenal cap where there was tenderness on palpation. A gastric analysis revealed 50 points of free acid and a total acidity of 80. The patient failed to obtain complete relief on medical treatment and was therefore transferred to the Surgical Service on April 23.

The old midline incision was reopened. The marginal ulcer at the anastomosis of the jejunum and stomach was adherent to the colon and an inflammatory reaction had already extended into the colonic wall. This was freed, and after considerable difficulty the old posterior gastroenterostomy was unhitched. The ulcer-bearing area was then excised and an end-to-end anastomosis made between the jejunal ends thus freed. A subtotal gastrectomy was then performed leaving only about 4 cm. of the upper end of the stomach, and a fresh loop of jejunum was anastomosed to the anterior wall of the remaining stomach. The entire procedure was beset with many technical difficulties and consumed four hours, but the patient left the operating room in fairly good condition. Twenty days after the operation a gastric analysis gave a free acidity of 12, a total acidity of 29. On the twenty-eighth day postoperative, he began to complain of pain deep in the epigastrium coming on late at night, and it was soon noted that it was relieved by food. A gastric analysis on the thirty-third day after the operation showed a free acidity of 56 and a total acidity of 66. He was started on a Sippy régime with a soft-solid diet. This relieved his discomfort during the day and an occasional pain at night was relieved by milk. A gastric analysis forty days postoperative showed a free acidity of 35 and a total acidity of 43. An x-ray made forty-one days postoperative showed only 5 cm. of the stomach remaining and a well-functioning anastomosis. No evidence of ulcer could be seen.

On August 1, 1932, approximately three months after his previous operation, the patient was seized with a severe abdominal pain which doubled him up. He continued to be so uncomfortable that he was again readmitted to the medical wards on August 5. X-rays showed the barium to be emptying rapidly and well into both jejunal loops. There was a smooth projection between the two loops containing a small residue of barium at six hours. The efferent loop was dilated but showed increased peristalsis. The cecum was irregular at six hours. Impression was a postoperative stomach, possibly a jejunal ulcer.

Further studies on the ward revealed the presence of a gastro-colic fistula. A gastric analysis at this time showed a free acidity of 78 and a total acidity of 93. The patient was again transferred to the Surgical Service where the fistula was excised and the ends of the intestine brought together. This operation proved too much and the patient died two days later.

There are certain factors common to all four cases. Each patient had a high gastric acidity. Although the original gastric analysis in Case I showed a free acidity of 45, a later analysis revealed an acidity of 101 after a gastroenterostomy had been performed, so that it seems probable that the original analysis was in error. Each case was treated by increasingly radical procedures. Each patient is worse today than when he was first seen.

In view of these results, the effect which surgery had upon the gastric acidity becomes of interest. Removal of the antrum, such as was done in Case I and Case II, did not lower the acidity and may have increased it. This frequently occurs following the operative procedures advocated by Polya, and Billroth in his second operation. These operations not only fail to reduce the acidity but they remove a portion of the stomach which normally produces an alkaline secretion; therefore, they should theoretically do more harm than good.

In Cases III and IV, a subtotal gastrectomy was accomplished. Only enough of the stomach was left to permit the suturing of the stomach

to the small intestine, yet, within a few weeks of the operation the acidity in both cases was as high as before the operation was performed. This agrees with Fauley's[2] finding that the stomach tends to hypertrophy after operations of this kind. If these radical operations do not effect a very decided decrease in gastric acidity, there is little reason to expect a cure; for the recent laboratory studies[3] strongly suggest that ulcers are dependent upon the damaging effect of the hydrochloric acid.

In looking back at the history of these four cases, it becomes evident that they would have been better off if medical treatment had been persisted in. Although the results with medicine would not be classified as satisfactory, yet the patients would be no worse today than at the time treatment was started. All four of these patients were engaged in active work when under medical treatment; today, one of them is dead; one has worked only at intervals during the last five years; one has not worked for four years and one has not worked for six years. It, therefore, becomes important to know when operations of this kind may be used with safety.

For a number of years I have felt that a patient showing a high gastric acidity and evidences of nervous instability was prone to develop jejunal ulcers after gastroenterostomy and have argued against its use in this type of case. Jejunal ulcers have always proved to be serious complications in our hands. Excision of the ulcer has been followed by the development of another if the enterostomy has been allowed to remain. Healing or controlling by medical means has proved more difficult than treatment of the original ulcer. For these reasons and because of the satisfactory results reported by others[4] with subtotal gastrectomies, the removal of most of the stomach seemed indicated in those cases which developed jejunal ulcers. However, our results show that this radical procedure cannot be relied upon to cure these unfortunate persons.

I have concluded, therefore, that when a patient develops a jejunal ulcer following a gastroenterostomy, an attempt should be made to heal it by medical means. If this proves unsatisfactory, the best form of treatment consists in undoing the gastroenterostomy and treating the patient by good medical therapy. It is better to subject the patient to a continuously stringent régime than to run the risk of disabling him with surgery.

As already stated, a certain group of patients with a high acidity and certain nervous characteristics are prone to develop jejunal ulcers. These patients are usually thin, flush easily, have moist palms and respond poorly to alkaline therapy. They frequently develop alkalosis on a Sippy régime and respond to alkalies by an increased flow of gastric juice. The failure of these patients to do well is not entirely dependent upon the high acidity, as the results of a large series of patients are much the same irrespective of the gastric acidity. (See table I.) Some other factor must come into play. A high acidity may be due to a failure of normal neutralization or to such an overproduction of gastric juice that the normal mechanism of neutralization is inadequate.

TABLE I

NUMBER OF SATISFACTORY RESULTS VERSUS THE GASTRIC ACIDITY

| Free Acid cc. n/10 per 100 cc. | No. Sat. Results | Total No. Patients Treated | % Sat. Results |
|---|---|---|---|
| 0-20 | 64 | 85 | 75.2 |
| 21-40 | 134 | 204 | 65.6 |
| 41-60 | 197 | 254 | 77.5 |
| 61-80 | 125 | 162 | 77.1 |
| 81 plus | 89 | 117 | 76.0 |

It is the latter type of case which is more difficult to treat medically and which shows a greater tendency to develop jejunal ulcers. This type is also prone to secrete acid long after the stimulus, in the nature of food, has disappeared. Early in the use of his method of treatment, Sippy recognized the importance of a continued secretion at night as a serious complicating factor in the way of therapy. Failure to recognize and check this continuous secretion is the most common reason for poor results with this treatment. Recently, Henning and Norpoth[5] have shown that this continuous night secretion does not occur in healthy persons. This is a complication of ulcers to which too little attention has been paid. It can be easily demonstrated that control of the acidity throughout the day is not sufficient for healing if a continuous secretion occurs at night. If an operation is performed where this condition is present, and the mucous membrane of the jejunum is forced to come into contact with the gastric acidity daily from 18 to 24 hours, it is unlikely that the resistance of the membrane will be sufficient to prevent the development of an ulcer. Therefore one should not advocate surgery in the presence of a continuous secretion. The presence or absence of a night secretion can be easily determined by passing a Levine tube through the nose and allowing it to remain in the stomach throughout the evening and night if necessary. Fortunately, the complication of a hypersecretion with obstruction is rather uncommon but if the two are present and surgery is required because of the obstruction, a pyloroplasty according to the Finney or Horsley type should be preferred. These two operations allow the mucous membrane of the duodenum, which is best suited to resist the corrosive ac-

tion of the juice, to receive the highly acid secretion. Once the obstruction is overcome, it is better to treat the patient medically even though some symptoms persist than to attempt complete relief through radical surgery.

### SUMMARY AND CONCLUSIONS

Four cases of duodenal ulcer are reported which were operated upon because of a failure to obtain complete relief by medical measures. Owing to the development of jejunal ulcers in three and a tendency to bleed in one, more radical procedures were attempted, resulting, finally, in the removal of from three-fourths to seven-eighths of the stomach. In no case has the condition of the patient been improved by this radical type of treatment.

The cases illustrate that there are a certain number of patients suffering from peptic ulcer which cannot be cured even by subtotal resection of the stomach and may, in fact, be made worse by it as these patients have been. Caution should, therefore, be used in deciding upon this operation. It is suggested that where a jejunal ulcer develops with the simpler operation such as gastroenterostomy, it is wiser to undo the enterostomy than to attempt more radical surgery. It is also suggested that poor results are more likely to occur where a continuous night secretion exists.

### REFERENCES

1 Emery, E. S., and Monroe, R. T.: To be published.
2 Fauley, G. B.; Strauss, H. A., and Ivy, A. C.: Studies on the effects of subtotal gastric resection in the dog. Am. J. Surg. 17: 427 (Sept.) 1932.
3 Dragstedt, L. B., and Vaughn, A. M.: Gastric ulcer studies. Arch. Surg. 8: 791 (May) 1924.
Gallagher, W. J.: Effects of injections of acid and trauma on jejunal transplants to the stomach. Arch. Surg. 17: 279 (Aug.) 1928.
Morton, C. B.: Observations on peptic ulcer. Ann. Surg. 85: 203 (Feb.) 1927.
Mann, F. C., and Williamson, C. S.: The experimental production of peptic ulcer. Ann. Surg. 77: 409 (Apr.) 1923.
McCann, J. C.: Experimental peptic ulcer. Arch. Surg. 19[1]: 600 (Oct.) 1929.
4 Berg, A. A.: The mortality and late results of subtotal gastrectomy for the radical cure of gastric and duodenal ulcer. Ann. Surg. 92: 340 (Sept.) 1930.
5 Henning, N., and Norpoth, L.: Investigations on secretory function of stomach during night sleep. Arch. f. Verdauungskr. 53: 64 (Jan.) 1933.

## LEUCOCYTOSIS IN MENTAL DISEASE*

BY JACOB KASANIN, M.D.†

THE normal variation of the number of leucocytes in peripheral circulation is usually given between 5,000 and 10,000. Although this figure is still adhered to by most textbooks, a great deal of recent work has brought out some facts which will probably modify our idea of the limits of physiological variation of the number of leucocytes in blood.

Thus Sabin[1], as a result of an extensive review of literature and her own investigations, stated that variation in the number of white cells in circulation may be over 100 per cent, pointing out that "for each individual the highest number of cells in any day tends to be twice the lowest number." She postulated a certain rhythm of the white blood cells just as there is a rhythm of the temperature of the human body. Sabin pointed out that the variation in the number of cells was much greater than the accepted values. Subsequent work by other investigators has confirmed Sabin's conclusions.

Thus Medlar[2], studying 17 healthy adults, came to the conclusion that a variation of 100 per cent in the total leucocyte count and 30 per cent in the differential may be found in an average healthy individual in the course of weeks without any clinical manifestations of disease. Christianna Smith[3] made the same observation in a group of young, healthy students in a girls' college. All observers agree that there is a rise in leucocyte count in the afternoon and that this rise is not necessarily due to digestion, because the same rise takes place when no food is given at noon. These investigators also found that the counts went as high as 13,000 without any evidence of disease. The exact mechanism of this wide fluctuation in the white count was not understood until certain theories were advanced first by Sabin and subsequently by Garrey[4].

In an explanation of this wide variation Sabin used a very graphic description of "showers" of cells being discharged into the peripheral circulation at definite intervals approximating an hour, thus imparting the idea of a certain rhythm in the discharge of the leucocytes. On the other hand, Garrey maintains that the leucocyte count remains very stable in the same individual as long as that individual is in a state of absolute rest. This he calls a "basal band", and he finds that under the condition of absolute rest the count varies between 5,000 and 6,000. On the other hand when the individual is normally active the count varies between 8,000 and 10,000, and 10 per cent show even a higher level. This is called by Garrey "the count at activity level". The same investigator believes that the high white counts are due to the fact that the patients have been more active than usual and in confirmation of this theory he brings up a large number of interesting observations. Thus a mild degree of exercise induced

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by a few knee bends may raise the leucocyte count to 12,000 or 13,000. A 100-yard dash raises a count from the normal activity level of 8,000 to 22,000 within 15 seconds. A subsequent 440-yard dash raised the count to 26,000. A competitive quarter-mile race caused a rise to 35,000. After a 45-minutes' rest the count came down to 30,000. A 5-minute oral quiz raised the count from 5,000 to 9,000 and 10,000. Garrey especially emphasizes the importance of nervous and psychic factors. Thus a sudden surprise by stimulation of the skin causes a rise in the count of 2,000 cells. When a hyperthyroid patient was told that he would have to be operated on, he showed a rise in his count from 7,000 to 18,000. In highly nervous cases the mere suggestion of blood-taking is sufficient to raise the count very promptly and significantly and might suggest a possible pathological leucocytosis. In his own laboratory Garrey[5] is extremely careful to reduce to a minimum the emotional stimulation in determining the basal count: thus the patient remains in bed and an especially sharp cataract knife is used so as not to cause any pain when puncturing the finger for the drop of blood. The pain of angina pectoris, in Garrey's observation, caused a leucocyte count of 18,000 although the basal level of the same individual was approximately 7,000.

In the field of mental diseases it was observed for a long time that many patients have shown high white counts and this was never explained except on the basis of possible infection, which, however, could not be demonstrated. The first large series of cases showing that there is a definite increase in the number of white cells in mental patients was done by Bowman[6], who in a series of 1,657 patients showed that in 50 per cent of these cases the white count was over 10,000 cells. Thus in 789 cases of schizophrenia 54 per cent of the cases had a white count above 10,000, 18 per cent of the cases had a count above 12,000, and 11 per cent of the cases had a count above 15,000. The same proportion held true for the cases of the manic-depressive type. In a group of 587 cases of affective disorder 53 per cent of the cases had a white count above 10,000, 21 per cent had a white count above 12,000, and 12 per cent of the cases had a white count above 15,000. In the 281 cases of general paralysis the proportions were slightly lower.

These extraordinary findings by Bowman attracted my attention. In an attempt to find a possible explanation for the high white count in so many cases, I took the figures obtained for all the new admissions to the Boston Psychopathic Hospital in 1927. During that period 1,553 patients were admitted and counts were made in all but 46 cases. Most of the counts were made in the afternoon by several technicians whose jobs consisted in doing the routine laboratory work of the hospital. In this series (See table I), only 45 per cent had a white count below 10,000, whereas 52 per cent of the cases had a white count over 10,000, and of these 18 per cent had a count between 12,000 and 15,000, 10 per cent had a count of 15,000 to 20,000, and 4 per cent of the cases had a white blood count of over 20,000. In order to find out the possible significance of these high white counts I took 100 cases who had a white blood count above 15,000 and compared them with 100 consecutive cases which had a white count below 10,000. Statistical tables were made showing the comparative figures of these two groups of cases in reference to positive physical findings, temperature, mood, activity, clinical diagnosis, sex, and age. I might say that most of these white counts were done within a few hours after the admission of the patients to the hospital and always within the first 24 hours after their admission. They were usually taken in the afternoon, either before, during, or after supper. A review of the various tables showed that in respect to physical findings the cases with a leucocyte count above 15,000 showed more evidence of infections, metabolic disorders, and also various conditions such as pregnancy, lactation, and puerperium. There were more cases below 10,000 which showed negative conditions, healing surgical wounds, or mild neurological signs such as unequal reflexes, facial asymmetry, and so forth. There were more cases with a rectal temperature above 100° in the group which had a white count above 15,000. Going over the mood of the patients I found that patients who were depressed, stuporous or elated, and whose mood was otherwise variable, showed more cases with a white count above 15,000, whereas the patients who were either apathetic, normal, or somewhat tense tended to have the white count below 10,000. The tables for age and sex did not show anything significant statistically. In the table showing distribution of cases by diagnosis there was a slight suggestion that cases showing the affective disorders had more frequently a count of over 15,000. All the above findings which I discussed were more suggestive than conclusive.

TABLE I

LEUCOCYTE COUNT

General Distribution in 1,553 Cases, All Diagnoses, Male and Female, Admitted to the Boston Psychopathic Hospital in 1927

| White Count | Number of Cases | Per Cent |
|---|---|---|
| Under 5,000 | 14 | 1 |
| 5,000-9,999 | 682 | 44 |
| 10,000-11,999 | 316 | 20 |
| 12,000-14,999 | 280 | 18 |
| 15,000-19,999 | 158 | 10 |
| 20,000- | 57 | 4 |
| Unknown | 46 | 3 |
| Total Number of Cases | 1,553 | 100 |

One of the tables, however (table II), suggested that there was a possible correlation between the leucocytosis and the amount of activity of the patients on the ward. In this table we can see that 58 patients who were either restless, overactive, or variable showed a white count of over 15,000, whereas 65 patients who were quiet, underactive, or stuporous showed a white count below 10,000.

The number of variables in mental patients is so great that it is impossible to study just one variable, but it is important to remember that these blood counts were done on patients who were in the hospital less than a day and who came there in the acute stage of their illness when they were still excited, upset, tense, unhappy, and very frequently distraught, overactive, and excited. This condition certainly existed in more than 50 per cent of the cases, and it is not surprising that we find blood counts of over 10,000 in a similar proportion of cases, which goes well with the recent work on the influence of emotional states and muscular exertion on the white blood count. The only table which was at all suggestive of a possible clue to the high white count was the one on activity of the patients in the hospital. Of course the question can be raised as to whether all these patients were not suffering from some physical disease, and the psychiatrist is usually accused of being woefully ignorant of the possible somatic and toxic factors which might be the cause of these psychoses. There is no evidence, however, that the patients with the high white count showed a great deal more illness than those that had the count below 10,000. Both groups stayed in the hospital the usual period of time and then went on to other hospitals without showing much significant difference between the two groups.

This paper is not an attempt to prove that a high white blood count does not mean anything in mental disease; it merely calls attention to the psychomotor state of the patient as a possible factor in the causation of the leucocytosis when all other factors have been eliminated and have been found to be negative. The Schilling index and the nuclear index were studied in great detail by Fleming[7] in a large series of cases and as a result of his study he became convinced that there must be some toxic or infectious factor in at least one-half of the cases of chronic mental illness, but physical and laboratory examinations in our cases did not substantiate that evidence. It is rather interesting that the white count shows more of the emergency reaction in mental cases than does the blood sugar. Thus in a large group of mental cases which I have studied with Dr. Bowman[8], we found no evidence that the blood sugar was any higher in emotional states in mental patients than it was in states of rest. It is interesting to note that when one compares the figures in an acute mental hospital with those in a chronic mental hospital, we find that in the latter the white blood counts do not tend to run so high, as I intend to show in a later communication. It is of note that in most cases showing leucocytosis of unknown origin the count tends to drop within a few days after the patient's admission to the hospital, as they get acclimated to the new environment and as they themselves quiet down to a more basal level of their activity.

TABLE II

THE WHITE BLOOD COUNT AND STATE OF ACTIVITY IN 200 CASES OF MENTAL DISEASES

| | Stuporous | Underactive | Quiet | Restless | Overactive | Variable | Total |
|---|---|---|---|---|---|---|---|
| W. B. C. 15,000 or above | 5 | 13 | 24 | 31 | 24 | 3 | 100 |
| W. B. C. below 10,000 | 1 | 10 | 54 | 21 | 14 | 0 | 100 |
| Total | 6 | 23 | 78 | 52 | 38 | 3 | 200 |
| Remarks | Includes semi-stuporous | | Includes normal and fairly quiet | Includes active | Includes excited, violent and tense | Overactive and underactive | |

## CONCLUSIONS

1. In a sample of 1,553 unselected cases of mental disease more than 50 per cent of the cases showed a white count of over 10,000 cells, and 14 per cent in the same group showed a leucocytosis of over 15,000 cells.

2. A group of 100 consecutive unselected cases with normal white counts (below 10,000) was compared with a similar group of 100 cases showing a definite leucocytosis (above 15,000). The two groups were compared with reference to sex, age, various physical factors, temperature, mood, diagnosis, and the amount of activity on the wards.

3. There were no significant statistical differences between the two groups. There was a possible suggestion, however, that overactivity was associated with the high white counts, while underactivity was associated with normal white counts.

4. It is suggested that in the absence of definite infection, toxicity, and other somatic fac-

tors the psychomotor state of the patients characterized by restlessness, tension, and overactivity may be responsible for the high white counts in such cases.

REFERENCES

1 Sabin, Florence R.; Cunningham, R. S.; Doan, C. A., and Kindwall, J. A.: The normal rhythm of the white blood cells. J. H. H. B. 37: 14 (July) 1925.

2 Medlar E. M.: The extent of the variations in the leukocytes of normal individuals. Am. J. Med. Sc. 177: 72 (January) 1929.

3 Smith, Christianna, and McDowell, Anna Mary: Normal rhythm of white blood cells in women. Arch. Int. Med. 43: 68 (January) 1929.

4 Garrey, W. E., and Butler, Virginia: The basal leucocyte count and physiological leucocytosis. Proc. Staff Meet. Mayo Clin. 4: 157 (May 15) 1929.
Ibid.: Physiological leucocytosis. Am. J. Physiol. 90: 355 (October) 1929.

5 Garrey, W. E.: Personal communication.

6 Bowman, Karl M., and Raymond, Alice F.: Physical findings in schizophrenia. Am. J. Psychiat. 8: 901, 1929.

7 Fleming, G. W. T. H.: Some aspects of the leukocyte associated with mental disorder. J. Ment. Sc. 78: 129, 1932.

8 Bowman, Karl M., and Kasanin, Jacob: The sugar content of the blood in emotional states. Arch. Neurol. & Psychiat. 21: 342 (February) 1929.

# DR. LUCINDA SUSANNAH (CAPEN) HALL

## The First Woman to Receive a Medical Degree From a New England Institution

BY FREDERICK C. WAITE, PH.D.*

THE first woman to receive a medical degree in the United States had no contact with New England. Dr. Elizabeth Blackwell was born in England in 1821, lived in New York and New Jersey from 1832 to 1838, in Cincinnati from 1838 to 1844, taught school in Kentucky and South Carolina, attended two courses at the Medical Institution of Geneva (N. Y.) College and graduated there January 23, 1849. She practiced in New York City from 1850 to 1869 and thereafter in London, England until her death in 1910.

In 1850 at Central Medical College of Rochester, N. Y. one woman was graduated. She was born in Massachusetts, but was not a resident of New England after 1844.

In February, 1851, at the same institution two women were graduated, one of whom was a native of Vermont, but a resident of New York after 1844. On March 6, 1851 two women were graduated at the Syracuse (N. Y.) Medical College, neither of whom had any relation to New England. In the closing days of 1851 the Female Medical College of Philadelphia graduated its first class. Of the eight women members one (Dr. Martha A. Sawin) was a native and resident of New England and returned to Boston to practice.

In February and March 1852 two medical schools at Cleveland, Ohio graduated each one woman. One of these (Dr. Nancy E. Clark) was a native and resident of New England and returned to Boston to practice.

On February 19, 1852 the Central Medical College graduated three women, one of whom (Dr. Parmelia R. Bronson) was a native and resident of Connecticut and returned to that state to practice. On May 27, 1852 one woman was graduated at the same institution. She was a native of England and for a time a resident of Massachusetts, but not after 1845. Diligent search has failed to disclose the names of the graduates in 1852 at Syracuse Medical College so that no statement can be made as to the number of women, if any, graduated there in that year.

Before June, 1852, twenty women had been graduated in medicine by six medical schools, namely Geneva, Central, Syracuse, Female Medical of Philadelphia, Western Homeopathic, and Western Reserve. Five of these women were natives of New England. Of these five natives three returned to New England to practice.

This sketch concerns a woman who was born in New England, spent all her life there, graduated from a medical school in New England, and practiced there for thirty-eight years. She was the first woman physician who in all three factors of birth, residence, and medical education was completely a New England product.

Lucinda Susannah Capen was born July 13, 1815, on a farm near Stewartstown, Coos County, N. H., a small hamlet among the hills in the extreme northwestern part of the state. She was the fifth child and third daughter of Ebenezer and Abigail (Carter) Capen, who, following their marriage in 1805, had gone from Concord, N. H. to Stewartstown in 1806. When she was about ten years old the family returned to Concord. Here she received a common school education.

At the age of eighteen on June 4, 1833 she was married by Rev. Nathaniel Bouton, long the pastor of the North Congregational Church of Concord, of which Miss Capen was a member, to Robert Hall, five years her senior and native on a neighboring farm. He was the son of Captain James and Ruth (Abbott) Hall, a daughter of Benjamin Abbott who was in the battle of Bunker Hill.

Mr. and Mrs. Robert Hall resided first at Loudon, N. H., a few miles east of Concord, for about two years, and then returned to Concord where they lived seven years. In Concord were

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born their only children, Ann Louesa on June 21, 1834 and Tilton Clark on July 7, 1839.

About 1842 Robert Hall became a lay assistant to Dr. Lemuel W. Paige (1807-1857) of the neighboring town of South Weare. The Hall family moved to that town where they resided for several years, returning to Concord before 1847. This physician, besides having an extensive practice, was engaged in the manufacture of certain favorite prescriptions, chiefly of an herbal nature, which were sold both through druggists and also along the countryside of northern New Hampshire and Vermont. Robert Hall from boyhood had been interested in botany so that this occupation was to his taste. In 1847 he is designated as an apothecary in Concord.

Although Mrs. Hall's maternal uncle (Dr. Thomas Carter) was a physician, having attended the Dartmouth Medical School in 1813, there is no record that, up to the time of going to South Weare, Mrs. Hall had any idea of studying medicine.

At South Weare she was given the privileges of Dr. Paige's library. The reading of his medical books aroused in her a keen desire to become a physician and under Dr. Paige's direction she pursued an orderly course of medical reading. However, there was no avenue by which she could attain her ambition to take a course in a medical school, for there then was no institution where women could study medicine. This was at least two years before Elizabeth Blackwell was admitted to the Medical Institution of Geneva College and before Harriet K. Hunt applied for admission to Harvard Medcal School.

However, a partial solution was about to develop. In 1845 Dr. Samuel Gregory of Boston began to agitate the question of medical education for women. He published two pamphlets arguing that trained women physicians should be available to treat the diseases of women. He was especially opposed to obstetrical practice by men, or what, both in Great Britain and the United States, were termed "men midwives". As a result of Dr. Gregory's efforts, in October 1848, was organized in Boston the Female Medical Education Society, which sponsored a course of instruction in midwifery which they called the Boston Female Medical College. This society had no charter at the outset. It could grant no degrees, but after the successful completion of one, or usually two, twelve-week terms of instruction the society issued a certificate of proficiency in midwifery.

The staff of the "college" consisted of three men. Dr. Gregory was administrative secretary, but gave no instruction. Dr. William Mason Cornell (A.B. 1827 Brown, M.D. 1844 Berkshire) who was both Baptist clergyman and physician, gave some instruction in materia medica and elementary medicine. The major part of the teaching was by Enoch Carter Rolfe (M.D. 1838 Bowdoin) who taught anatomy, physiology, and midwifery. His share was so great that the institution was frequently called Dr. Rolfe's school. Both Dr. Cornell and Dr. Rolfe were in good standing in the profession and Fellows of the Massachusetts Medical Society.

The first course began November 1, 1848 with a class of twelve women of whom Mrs. Lucinda Hall was one. Mrs. Hall, during this course lived at the home of a physician of East Boston who was a relative. Due to her previous study under Dr. Paige she was able to attain the certificate of proficiency at the end of the first course, and was a member of the first small class of "graduates" of this "college," consisting probably of four women. She was at this time thirty-three years of age and her youngest child was nine years old. Mrs. Hall returned to Concord, N. H. where she practiced midwifery. For several years she was the only woman in New Hampshire holding the certificate of this institution.

However, this attainment did not satisfy her ambition. She had received no instruction in surgery in the course at Boston, and little in medicine, and her desire was to take a full medical course and attain a degree in medicine. At this point enters the physician of East Boston in whose family she had lived in 1848.

In 1850 the Central Medical College of Rochchester, N. Y. had instituted a female department. This was an eclectic school. In Worcester, Mass. was another eclectic medical school organized in 1846 and recipient, in 1849, of a Massachusetts charter. Late in 1850 Calvin Newton, the dean of the Worcester Medical Institution wrote that he had been solicited to establish a female department, but no decision had been reached. The physician in East Boston repeatedly urged upon Dr. Newton that Mrs. Hall be admitted to the medical school and in March, 1852 three women were admitted to the Worcester Medical Institution. One of these three was Mrs. Lucinda Hall.

The requirement for graduation in this school was the conventional three years of medical study and attendance on two courses of medical lectures. It is evident that Mrs. Hall's study under Dr. Paige was accepted as a year of medical study and also that her course of instruction at the Boston Female Medical College was accepted as one course of medical lectures, since she attended but one course at Worcester, that extending from early March to late June, 1852.

That she was in Worcester previous to the opening of the course is shown by her advertisement in the Worcester Spy of February 7, 1852 soliciting midwifery cases and stating that she could exhibit the certificate of proficiency issued by the Boston Female Medical College. Her

name does not appear in the Worcester City Directory at any time. Moreover, that her sojourn in Worcester was considered temporary is shown by the fact that both she and her husband, who entered the school at the same time, are listed in the medical school catalogue as from Concord, N. H.

On June 23, 1852 Lucinda Hall, at the age of thirty-seven, received from the Worcester Medical Institution the first medical degree conferred on a woman by a New England institution. The New England Female Medical College did not confer its first medical degrees until 1854 when four women were graduated.

Neither of the two other women who attended at Worcester in 1852 were graduated with Dr. Hall. Extensive search has failed to discover the names of the graduates of this school in the following year of 1853 when it is possible that they graduated. The names of these two other women, have not been found in the lists of graduates of any other medical school of this period. In October, 1853, shortly after the death of Doctor Newton, the trustees of the Worcester Medical Institution voted to cease accepting women as students, but in 1856 again accepted them and graduated women in the classes of 1856 and 1857. The school probably closed in June, 1857, but it is possible that it gave one more course in the winter of 1858-1859.

Dr. Robert Hall, in published reminiscences, states that his wife evolved the idea of becoming a physician before he did. However, he entered the Worcester Medical Institution at the same time as his wife, but this being the beginning of his formal medical study, he did not receive his degree until June, 1854, two years after his wife's graduation.

The professor of surgery at the Worcester school when Dr. Lucinda Hall graduated was Walter Burnham (M.D. 1829, University of Vermont). He had attained a considerable reputation as a skilful surgeon and was then in practice in Lowell, Mass. At his solicitation Dr. Hall settled for practice in Lowell immediately after her graduation. She was the fourth woman holding an M.D. degree to enter practice in New England, the three others already mentioned (Drs. Sawin, Clark, and Bronson) having preceded her by a few months, but all in the same year of 1852.

Dr. Hall's family took up a residence at 120 Merrimac Street, Lowell. When Dr. Robert Hall graduated in 1854 he joined his wife in medical practice. They developed a successful practice in Lowell, but late in 1856 double tragedy entered their home.

Their son, then seventeen years of age, while hunting near Concord, N. H. on October 11, 1856 was killed by the accidental discharge of his own gun. Their daughter at sixteen years of age, in 1850, had married and gone to Ohio. Here, within a few weeks after marriage, both she and her husband contracted cholera and her husband died. She returned to her parents' home. Early in 1856 she was married to Samuel Fletcher Bouton (1837-1902), a son of the Reverend Nathaniel Bouton already mentioned. They went to Chicago where he and his brothers were in business. Here on November 2, 1856, a son was born. Exactly two months later on January 2, 1857, Mrs. Bouton died.

The infant son had been named Tilton Clark Hall Bouton after his deceased uncle. The Drs. Hall took this grandson into their home and reared him. He graduated from Dartmouth College in 1878 and from Andover Theological Seminary in 1881. For a period of fifty years he held pastorates successively in five important towns of New Hampshire and Massachusetts, and is now retired. To Reverend Bouton the author is indebted for many facts concerning his grandparents that would otherwise have been missed.

Due to the domestic tragedy by which both of their children died within a period of three months, the Drs. Hall, early in 1857, returned to his boyhood home, near Concord, N. H. Here they practiced for nine years. Dr. Lucinda Hall was more enthusiastic about practice than was her husband. For several years she was the only graduated woman physician in New Hampshire. Her practice was chiefly in obstetrics and diseases of women and was more than local, since at times she returned to her old patients in Lowell and occasionally had patients in Boston.

Early in 1866 the Drs. Hall bought a tract of land in the western part of Concord containing the Birchdale Mineral Springs and built a residence. This was followed by the erection of a sanitarium. It was not a water-cure, a type of institution much in vogue in the northeastern part of the country from 1835, but rather a sanitarium for convalescents and chronic cases, and had a capacity of about fifty patients. This building, known as the Birchdale Hotel, was opened in 1867, and continued as a popular health resort until it was burned on July 26, 1885 with a loss of $25,000. The Drs. Hall disposed of the sanitarium in 1876, but continued to occupy the residence until 1892.

About 1866 Dr. Lucinda Hall developed respiratory disease which extended to pulmonary hemorrhages. Due to her frail health, during the next ten years she restricted her practice largely to the patients of the sanitarium, but continued to accept obstetrical cases in other places especially in Lowell and Boston. Also at times she visited professionally former patients of the sanitarium who had gone to their homes.

After retiring from the sanitarium in 1876, because of frail health, she restricted her practice to former patients and old friends. She continued this limited practice until a few days before her death which occurred at Birchdale, Concord, N. H., on August 27, 1890, at an age past seventy-five years. The immediate cause of her death was cerebral hemorrhage, a first onset being followed in ten days by a second. She is buried in the Millville Cemetery three miles west of the statehouse in Concord.

From newspaper notices at the time of her death one learns of the high regard in which she was held in the community, both professionally and personally. From other sources it is learned that she was always enthusiastic about her profession, and in return her clientele were grateful for her professional service. Her initiative, from which developed her eager desire to become a physician, sustained her through the difficulties which in those days impeded the path of a woman who wished to enter the profession. Having once entered practice her enthusiasm, industry, ability, and personality carried her on in successful medical service in spite of family sorrows and frail health. Her career is a fitting one for the pioneer of those many New England women who have entered the medical profession since Dr. Lucinda Hall received her degree nearly eighty-two years ago.

Dr. Robert Hall was interested in public service as well as in his profession. He held several minor appointive and elective public offices and in 1867 and again in 1868 was a member of the New Hampshire legislature. He died of cancer at Concord, N. H. on January 10, 1902 at an age past ninety-one years. Extensive newspaper notices at the time show that he had the respect and confidence of the community both for his professional and his public services.

The entries in medical school catalogues, show that many, possibly a majority of the early women medical students, were married when they entered medical school. One finds many cases where the wife of a physician followed him into the profession and practiced with him, but this is the only case that the author has found where the order is reversed and the wife graduated first to be followed into the profession by her husband.

This sketch of professional and domestic episodes in the life of a member of the first class of the Boston Female Medical College, who was also the first woman to receive a medical degree from a New England institution, serves to show to the present-day medical student that great difficulties may be successfully overcome when initiative, perseverance and energy are applied to their solution.

## SCHOLARSHIP AVAILABLE

A full tuition scholarship of $500 is available in the field of health education, for 1934-35, at Massachusetts Institute of Technology through the Department of Biology and Public Health. This scholarship covers the full scholastic year, beginning in September and closing in June. It is available for women only.

The scholarship will be awarded to a candidate recommended by the National Tuberculosis Association. Applicants should have basic training in mathematics, physics, chemistry, and biology. Undergraduate training in psychology and education is desirable. The awards will be based upon the nature and quality of the previous academic work of the applicant, personality qualifications for professional work in the field of public health, and need of scholarship aid. Preference will be given to candidates who possess the Bachelor's degree, and have had successful teaching or administrative experience.

The scholarship will be awarded in June, 1934 and applications should be received not later than May 15. All those who are interested in this scholarship are invited to write to the Child Health Education Service of the National Tuberculosis Association, 450 Seventh Avenue, New York, for application blanks.—*Bulletin National Tuberculosis Association.*

## MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

### Division of Adult Hygiene

### Notice to Cancer Clinic Staff Members

The February *Bulletin* was mailed to all registered physicians in the State, with the request that those wishing to receive it regularly return an enclosed application blank. About one thousand requests have been received. The *Bulletin*, therefore, will hereafter be mailed to that number of physicians, in addition to those who have previously received it as members of the staffs of the Cancer Clinics. Whenever there are news items or notices of special interest to the staff members, these will precede the abstract of the month.

This month we announce that on April 11th, another Cancer Clinic will be conducted by the Cancer Committee of the Massachusetts Medical Society, for the special interest of those physicians who are actually working with malignant disease. A clinical session will be held at the Massachusetts General Hospital. Luncheon will be served at the Pondville Hospital followed by an afternoon clinical session. Details of these clinics will be mailed to staff members later. — Number 11, *Cancer Clinic Bulletin*, March 1, 1934.

# CASE RECORDS
## *of the*
# MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

## CASE 20121

### PRESENTATION OF CASE

*First admission.* A forty year old American paper mill worker entered complaining of shortness of breath of six weeks' duration.

Five years before admission the patient noticed moderate palpitation on exertion. Fifteen months before admission while working he had a choking, nauseated feeling with pain over the epigastrium. He stopped working for a few minutes until the attack was over and then continued. Since then he had had frequent similar attacks, usually occurring at night but frequently while sitting in the evening or while working about the house. The common characteristics of these attacks were apparently a choking sensation and pain in or just over the epigastrium. With the attacks there was usually nausea. The attacks varied in frequency, sometimes occurring every day for a week and at other times occurring about once every one or two weeks. Sodium bicarbonate gave some relief occasionally.

He visited the Outpatient Department of this hospital ten weeks before admission to the wards. He was given glyceryl nitrate, which gave him marked relief. He gradually became progressively dyspneic, even on the slightest exertion, developed a dry cough, and recently had become definitely orthopneic. Six days before admission his condition became worse and edema of his ankles appeared.

His family and past histories are irrelevant. There is no history of rheumatic fever, tonsillitis, chorea, or syphilis.

Physical examination showed a well developed and fairly well nourished man propped up in bed, anxious, restless, sweating, and coughing frequently. His mucous membranes were slightly pale. There was no cyanosis. There was visible pulsation of the radial and temporal arteries. There was no venous engorgement. The heart was markedly enlarged to the left. The apex impulse was in the sixth interspace outside the nipple line. There was a very questionable thrill over the base. The sounds were of poor quality. There was a distinct third sound heard at the apex and to the left of the sternum, giving a protodiastolic gallop, rate 104°. A systolic non-transmissible murmur was heard at the apex and a systolic at the aortic area, not transmitted to the vessels of the neck. There was a short blowing diastolic murmur heard best in the third left interspace at the edge of the sternum. There were frequent extrasystoles. No apical diastolic murmur was heard. The radial arteries were palpable. The pulse was collapsible. The blood pressure was 120/60 in both arms. There were many medium moist râles up to the midscapular region on both sides. The respirations were 35. The liver dullness was down to three or four fingerbreadths. There was a moderate amount of edema of the legs and sacrum. There was no clubbing of the fingers.

Examination of the urine showed a specific gravity of 1.022 and a trace of albumin. The blood showed a red cell count of 4,500,000, a hemoglobin of 95 per cent, a white cell count of 6,600 with 74 per cent polymorphonuclears. The stools were negative. The non-protein nitrogen was 32 milligrams. A Hinton test was negative.

An electrocardiogram showed premature ventricular beats interrupting the normal rhythm at a rate of 80 to 85. There was bundle branch block with left axis deviation. The P R interval was 0.21. $P_1$ was notched.

X-ray examination showed a considerably enlarged heart. The transverse diameter measured 17 centimeters, 5 on the right and 12 on the left. The transverse diameter of the chest was 29 centimeters. The oblique view demonstrated obliteration of the posterior mediastinum by enlargement of the left and right auricles. There was also prominence in the region of both ventricles. The aorta appeared to be of normal size. The lung fields were clear. The left costophrenic angle was shallow. The hilus shadows were prominent on both sides.

The patient remained in the ward for seventeen days and was discharged improved.

*History of interval.* He remained at home following his discharge, spent most of his time in bed and felt quite comfortable, with very little breathlessness. The afternoon of his readmission he suddenly began to have severe abdominal cramps which caused him to double up and cry out. There was no diarrhea or vomiting. He ate supper thinking it might relieve the pain. It became much worse, however. He was frightened, and shortly after the onset of the cramps became markedly dyspneic, broke out into a cold, profuse sweat and, according to his friends, became extremely pale. This abdominal pain continued until his admission to the Emergency Ward at about ten o'clock that night. He had no precordial pain or cough. He had no edema during the interval and only slight orthopnea.

*Second admission,* two weeks after his previous discharge.

Physical examination showed a man propped upright in bed in acute respiratory distress and complaining of abdominal pain. His face was

pale, cold and covered with perspiration. He was anxious and restless. The extremities were cold and deeply cyanotic. The skin on his body was purple and blotched. The veins of the neck were moderately distended. The heart was enlarged to the left and right. The apex impulse was diffuse and feeble. The sounds were of fair quality. There was marked gallop rhythm, rate 120. No murmurs were heard. The blood pressure was not obtained. The posterior chest was not examined. Anteriorly no râles could be heard. The breath sounds were loud. The abdomen was full and tympanitic. The liver edge was not definitely felt, but there was resistance and dullness in the right upper quadrant three to four fingerbreadths below the costal margin. Rectal examination was not done. There was no edema.

Examination of the blood showed a red cell count of 5,400,000, a hemoglobin of 90 per cent, a white cell count of 28,500, with 83 per cent polymorphonuclears. One stool showed a two plus guaiac.

He was given a quarter grain of morphia on arrival and a venesection of 300 cubic centimeters was done. The breathing improved almost immediately and the pulse soon became stronger. He failed very rapidly however, and died on the second day following admission.

### Differential Diagnosis

Dr. Paul D. White: This case is one of much interest and difficulty. There are a few points in the history and examination on which I wish to comment.

In the first place he is a man of forty, just at the age when given heart disease we have a number of etiological possibilities. There are two chief problems in diagnosis, first, the kind of heart disease and its structural defects, and secondly, the terminal events associated with his severe abdominal pain.

His cardiovascular history begins at thirty-five. He had moderate palpitation on exertion. That most probably is effort syndrome; whether associated with heart disease or not we cannot say.

At the age of thirty-eight he began to have what should be interpreted as angina pectoris. That is two years before he died or fifteen months before his first hospital admission. These attacks were relieved by nitroglycerin. This was followed by presumable heart failure, beginning on the left side with dyspnea, and then going on to ankle edema and other indications of failure of the right heart.

The past history does not help us. There is no history of rheumatic fever, tonsillitis, chorea or syphilis.

There was visible pulsation in the radial and temporal arteries. Such increased pulsation is usually associated with hypertension or with aortic valve disease and aortic regurgitation. There was no venous engorgement, but at that particular time there was no evidence of congestive failure so far as the right ventricle was concerned. The heart was markedly enlarged to the left, so that some factor must have been present at that time, causing marked cardiac enlargement; such a factor could have been either mitral valve disease or coronary thrombosis. Angina pectoris with coronary disease does not give cardiac enlargement. Angina pectoris is frequently associated with a heart of ordinary size. We are not told whether the thrill at the base was systolic. It would be interesting to know what the findings were on physical examination at the time of discharge. Is there a note as to whether a thrill was palpated at the base?

Dr. Augustus S. Rose: Yes, sir. There was a thrill at the base at the time of discharge, thought to be systolic. Shortly after his admission the first time a definite diastolic murmur was heard at the base, interpreted by several observers as being a presystolic rumble, in addition to the other murmurs described.

Dr. White: These findings are of great importance. Prodiastolic gallop rhythm at the time he came in first, when he showed congestive failure, may be interpreted in a number of ways. The usual cause is dilatation of the left ventricle.

There is a statement that the diastolic murmur was heard along the left border of the sternum, indicating aortic regurgitation.

The blood pressure was normal; the pulse pressure, 60 with a systolic of 120, was less than one would expect, and yet not indicative of any high degree of aortic regurgitation.

There was no note of examination for penile scar?

Dr. Rose: It was looked for and not found. The Hinton test was negative.

Dr. White: The electrocardiogram one would like to see oneself, to determine how much bundle branch block there was. The degree is important. If there is a high degree one must consider that the coronary circulation probably had failed and that coronary thrombosis is likely; if only a slight degree, it may be associated with other than coronary disease. The P wave in Lead 1 is very important as evidence of mitral disease. If there is a well-marked $P_1$ wave and it is notched it generally means mitral stenosis.

The x-ray shows more indication of left ventricular enlargement than of right, probably because of the failure to find the characteristic prominence in the region of the pulmonary artery associated with mitral disease in contrast with an aortic valve lesion.

Dr. Tracy B. Mallory: Have you anything to add to that, Dr. Holmes?

Dr. George W. Holmes: No.

Dr. White: The aorta was of normal size; that is substantiated by the evidence you see

there. The lung hilus shadow was prominent on both sides; this is of some importance.

Finally this man had severe abdominal pain which was followed by death, and was attended by evidence of increased heart failure. The blood pressure was not obtained and murmurs which might have been evidence of a heart functioning well had disappeared.

The differential diagnosis must rest primarily between two conditions: (1) "rheumatic" heart disease with aortic stenosis and regurgitation and probably mitral valve disease, angina pectoris, and congestive failure, and (2) cardiovascular lues with aortic regurgitation, angina pectoris, and congestive failure. The functional diagnoses are easy. It is the etiological and structural changes that are not so easy. However, if it is certain that an aortic systolic thrill was felt and since we have evidence that the aorta was not dilated, that the Hinton reaction was negative, and that this man developed symptoms before he was forty, the diagnosis is preponderantly that of rheumatic heart disease, I should say at least eighty per cent probable. Aortic stenosis is a very important lesion and has been often overlooked in late years. It is not uncommon. It is almost as common as is mitral stenosis; we find this out when we look for it.

Cardiovascular lues must be considered as our second diagnostic choice, but way down the line. Subacute bacterial endocarditis with vegetation on the aortic valve would not be very likely because of the history dating back fifteen months and the attack after his first admission. Then there are other conditions of unknown etiology. On several occasions during the past few years we have been surprised by finding large hearts with no explanation of the enlargement. But here the diagnosis seems to me almost certainly that of rheumatic heart disease with aortic stenosis and regurgitation, mitral valve disease, and angina pectoris, which may mean an important degree of coronary disease, although we have also found it (angina pectoris) in middle-aged patients with aortic stenosis, which may cut down the coronary blood flow without the presence of much coronary disease.

Finally we come to the terminal illness, which so far as I can see is probably that of mesenteric embolism. There may be some other complicating surgical condition, but the cardiac enlargement suggests intracardiac thrombus formation with embolism as the terminal event.

Dr. Howard B. Sprague: When I saw this patient first I favored the luetic diagnosis because of the fact that he was discharged from the army in 1919 with a normal heart, because he had had two Neisser infections, and because he had had such a rapid downhill course. That was before his x-ray study. After finding that there was no dilatation of the aorta sufficient to account for the rather slight systolic thrill that one sometimes gets in luetic aortitis, and finding a generally enlarged heart, I felt that the diagnosis was probably rheumatic heart disease with aortic regurgitation and some stenosis, probably with mitral involvement, although the possibility of a complicating luetic aortitis might be considered.

Dr. White: Yes, one must consider the possibility of "rheumatic" aortic valve disease complicated by luetic aortitis, which would help to explain the angina pectoris and the electrocardiogram.

Dr. Sprague: The electrocardiogram showed definite bundle branch block. I thought that probably could be explained by some interference with the coronary ostia dependent upon the aortic valve disease.

Dr. Frederick T. Lord: Our diagnosis, as far as the heart was concerned, would coincide with Dr. White's, that is an aortic valve lesion and mitral involvement, but the latter would have to be considered as possibly functional, as the diastolic may be an Austin Flint murmur rather than an indication of actual involvement of the mitral valve. Angina pectoris seemed reasonable from the story. There was also congestive failure. The most troublesome aspect from the diagnostic point of view was the abdominal disturbance. The pain was low in the abdomen, colicky in nature, and associated with bloody stools. The complex suggests some abdominal condition. We considered congestive failure as a possible explanation; but it did not seem reasonable to think that congestive failure could account for the colicky abdominal pain, and mesenteric thrombosis seemed a reasonable conclusion.

As far as the lungs were concerned we did consider, because of the suggestion from the X-ray Department, that he might have an infarct or a bronchopneumonia. I should think however on looking at these films that one might ascribe the pulmonary disturbance to congestive failure.

### Clinical Diagnoses

Rheumatic heart disease.
Congestive failure.
Mitral stenosis.
Aortic stenosis and regurgitation.
Mesenteric thrombosis.

### Anatomic Diagnoses

Cardiac infarct.
Coronary sclerosis.
Chronic endocarditis of the aortic valve, with slight stenosis and regurgitation.
Fusion of posterior aortic cusps.
Cardiac hypertrophy and dilatation.
Peripheral edema.
Chronic passive congestion.

### Pathologic Discussion

Dr. Mallory: I am afraid that in this case I am not going to be able to answer a good many questions.

He had a greatly enlarged heart, weighing 750 grams, and the aortic valve showed an interadherence of two cusps with some degree of fibrous thickening of the edges pretty typical of a healed, inactive rheumatic aortic involvement, not of great severity however. It seemed as though that alone in no way accounted for the very large size of the heart. The mitral valve was entirely negative, a point which is distinctly against rheumatic etiology. The aorta was negative, so I think we can safely rule out lues.

There was a large area of infarction in the left ventricle on the posterior wall involving part of the interventricular septum. The central portion of this infarct was somewhat thin and fibrous, the peripheral portions gray and soft.

The coronary arteries showed perfectly normal orifices. It did not seem as though the valve involvement could have affected them in any way. There was definite sclerosis throughout all branches of both coronary arteries, but no thrombosis could be made out anywhere.

As is quite frequently the case in these very large hearts, we failed to find a coronary thrombus in association with the infarct. In a heart of nearly normal size I think we have never failed to find a thrombus if we examined it carefully.

Dr. White: How old was the infarct?

Dr. Mallory: I should say it was a matter of months.

We were limited in our examination by restrictions and could not make a complete examination of the abdomen. We simply went in through a hole made in the diaphragm. We were able however to examine the whole intestine externally and it was not discolored at any point, so I do not believe he had a mesenteric thrombosis. What his terminal bleeding in the gastro-intestinal tract was I cannot explain. I have never seen mesenteric thrombosis in which gross discoloration of the serosa of the small intestine was not present.

A Surgeon: Was there any free fluid in the abdomen?

Dr. Mallory: No; the surfaces were smooth and glistening throughout.

Dr. Holmes: Was there anything in the lung?

Dr. Mallory: No, it was negative except for chronic passive congestion.

Dr. White: I should like to ask about the attack of pain. Was it in the lower abdomen?

Dr. Rose: He described it as being chiefly in the lower abdomen, but it was generalized and we could not be definitely sure. I think it is fair to say he had abdominal pain, but I think it is better to say it was generalized with some in the lower abdomen. It certainly was colicky in nature, because as he was in bed in the ward he would suddenly cry out.

Dr. White: This is certainly a very unusual combination of valvular disease and coronary disease. It is something we almost never see in young patients. We rarely get coronary thrombosis under forty, but we must remember that it does occur; we have encountered about 15 cases in the past decade. In this case we doubtless should have paid more attention to the severe angina pectoris and to the bundle branch block by electrocardiogram.

Dr. Mallory: It is interesting to note that in the last case with twenty-five years of hypertension the heart was normal in size. In this case with a minimal aortic valve lesion, although a definite one, and no hypertension, we have a heart weighing 750 grams.

Dr. White: Was the infarct a large one?

Dr. Mallory: It was of fairly good size, 4 centimeters in diameter.

Dr. White: We were once taught that coronary disease does not give rise to enlargement of the heart, but now we know differently, for some of the largest hearts we have seen have developed after coronary thrombosis. There is no doubt about the reaction of the heart to a large myocardial infarct with considerable increase in heart weight. Here, however, we have a complication with another condition, namely aortic stenosis and regurgitation.

Later Note by Dr. White: On reviewing the history at its source I find clearer clinical evidence for the earlier coronary involvement. The very first severe attack of substernal and precordial pain a year or so before death is said to have lasted an hour or more. This tends to rule out ordinary angina pectoris in favor of actual coronary blocking (occlusion).

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## CASE 20122

### Presentation of Case

*First admission.* An American housewife twenty-nine years old entered the hospital for the first time with a history of slight vaginal bleeding of three days' duration.

The patient was first seen eleven weeks before entry. She gave a history of having been married eight weeks earlier and having had no regular periods since that time. At this visit she complained of unusual bleeding and nausea and vomiting. The bleeding began twenty-four days before the visit and continued until six days before it, at which time it increased in severity and amount, but was not really profuse. The history appeared to be consistent with a very

early miscarriage. In view of the fact that the bleeding had been going on for two weeks it was considered safe to make a vaginal examination. It showed a very tight introitus and the uterus not palpably enlarged, in normal position. The adnexa were not enlarged and were not particularly tender. She was advised to take one teaspoonful of ergot three times a day for four days and to return for an examination. Four days later she said that there had been quite free bleeding without clots for the first two days. The flowing had practically ceased. She continued to take ergot. She was seen again in six days. She said that the bleeding had decreased in amount, although it was still present. Bimanual examination showed the uterus to be apparently slightly enlarged. She still continued to have nausea and vomiting in the morning for which she was advised to eat frequent small carbohydrate meals. She was seen again two weeks after the third visit. The nausea and vomiting had persisted and there had been practically no bleeding. Examination showed the uterus to be definitely enlarged. The adnexa were negative. Three weeks later she complained of considerable nausea and vomiting. Bimanual examination showed the uterus to be enlarged to the size of a three and a half months' pregnancy. Early in the morning, two days before admission to the hospital, after intercourse the previous night, she had a small amount of red bleeding, and had similar bleeding on the following morning and again on the day of admission.

Family history. Her mother had twins.

Physical examination. On admission the uterus was enlarged to the size of a four months' pregnancy.

She was kept in bed and given sedatives. She continued to have considerable staining. Eight days after admission she was given morphia and scopolamin early in the afternoon. After they became effective she was given hourly two minim doses of infundin intramuscularly until 9.30 p. m. The following day she had only an occasional uterine contraction with a very small amount of bleeding. She was then given repeated two minim doses of pituitrin, and on the following day eight three minim doses of pituitrin. This did not produce any labor. Two weeks after admission she was discharged. The bleeding had practically stopped.

*History of interval.* Following her discharge she was seen at regular intervals. She continued to bleed a little from time to time, and on the day before admission began to have uterine contractions.

*Second admission.* She was readmitted eight weeks after her previous discharge in labor, with pains coming about every five minutes. Shortly after admission she was examined rectally bimanually with the escape of fluid and hydatid material. Fifteen minutes later she passed a very large quantity of mole and fluid, about 300 cubic centimeters. A dilatation and curettage was immediately performed. She was given pituitrin and ergot, one ampule each. The uterus shut down very satisfactorily without excessive bleeding. She made a normal convalescence and was discharged twelve days after admission.

## Clinical Discussion

Dr. Paul Gustafson: A vaginal examination was made at the first office visit because of the fact that bleeding had been going on for two weeks. Of course there is very little increase in size in the first month of pregnancy, so that the uterus did not seem unusual. The most likely diagnosis seemed to be an early incomplete miscarriage because of the early bleeding for some time, and because the uterus was not enlarged I felt we had nothing to expect and the patient was given fluid ergot. Later a diagnosis was made of possible continuing pregnancy because of the vomiting and nausea. In other words we backed away a little and thought that in spite of ergot and bleeding pregnancy might be continuing. I might add that this young woman was very nervous and one who might be expected to have a great deal of vomiting.

The fact that at the fourth office visit the uterus was enlarged to the size of a three and a half months' pregnancy led to the thought of multiple pregnancy or mole. We thought of the possibility of hydramnios, which is found early in pregnancy in cases of anencephalic monsters, etc.

You are wondering why we did not do something. We did not know positively that she was not normally pregnant.

"On admission the uterus was enlarged to the size of a four months' pregnancy". My impression was that the uterus had not grown since her fourth visit. Her last period was five and a half months earlier and the uterus was small, whereas a couple of months previously it had been large for the date.

The blood pressure was 150/90.

There is something which I did not know at the time but which she told me later. She noticed small masses coming away from the vagina that looked like grapes. She did not tell me because she did not want to bother me and wanted to wait until she had labor pains. She was examined bimanually and, on pressing on the fundus, material which grossly looked like hydatidiform mole was passed.

She was curetted under gas-oxygen anesthesia and a large amount of hydatidiform material was removed. The amount was estimated at 350 to 400 cubic centimeters. At the time of operation it was not found that the uterine wall was thin, as is reported in some of these cases. There was an unusually small amount of bleeding at the time of operation. These cases are sometimes reported to have an extremely

large amount of hemorrhage. It was not so in this case. We waited until the patient was really in good labor.

She made a perfectly normal convalescence and was discharged eleven days later in good condition.

The urine was sent to Dr. Albright's laboratory for an Aschheim-Zondek test which at that time was reported negative.

DR. TRACY B. MALLORY: Dr. Titus, will you discuss the case?

DR. R. S. TITUS: I think the most interesting thing about it is the fact that the bleeding occurred so early. It is unusual for the bleeding in a hydatid mole to start as early as this.

The next interesting feature is the absence of very unusual increase in the size of the uterus as time goes on. These moles oftentimes go way beyond three months and have been known to go six or seven months with tremendously big uteri.

The physical diagnosis, when one has courage enough to make it, is made on the abnormal increase in size of the uterus, which suggests it and makes one think it exists, but it usually is not absolute until one sees the discharge of grape-like bodies that are pathognomonic of the condition.

I am interested to know if Dr. Gustafson has seen the patient since she left the hospital.

DR. GUSTAFSON: This is a very recent case.

DR. TITUS: The association between this disease and chorioepithelioma always leads one to feel that the patient should be watched for six months or more afterward.

The next question that comes to my mind is whether the profession has changed its mind regarding the possibility of giving radium subsequent to curettage. These cases are not at all common. In private practice I have seen only two, and in one of these the diagnosis was made by the patient herself as much as by anybody else. She had a tremendous amount of bleeding that she described as watery and pink, and when she came into the hospital she was still bleeding a watery discharge; no grapes. When one put the speculum into the vagina, however, one could see the grapes coming through the cervix as well as on the cervix.

### DIAGNOSIS

Hydatid mole.

### PATHOLOGIC DISCUSSION

DR. MALLORY: The surgical specimen is exactly what one would expect. I would estimate its size somewhat higher. There is at least 500 cubic centimeters of material here, innumerable grape-like spherules.

The most interesting thing from the pathologic point of view is always the question of ruling out chorioepithelioma, the malignant form of the same tumor. Often the slides of these moles show a considerable degree of active proliferation, and it may be very difficult from the slide to decide whether or not the tumor is entirely benign. In this case we saw no reason to think it was not benign.

I am not sure that the gross appearance of the material is not more important than the microscopic examination for evidence of active proliferation. I think in a gross appearance like this, one can be quite reasonably safe in considering it benign.

One rather interesting point in regard to moles which has only recently received much attention is the frequency with which multiple lutein cysts develop. I have here the ovaries of another patient in whom each ovary contains a half dozen large lutein cysts.

DR. JOE VINCENT MEIGS: I should like to say a few words. Almost all the literature states that this ovarian condition is not an uncommon thing, that the moles must not be removed, and that if the mole or the chorioepithelioma is removed they retrogress. I think, however, that this case which Dr. Mallory has just mentioned gives a very different point of view, because there is no normal ovarian tissue left at all and it probably could not retrogress. I think that this is probably the same sort of response in the ovary that we get in using the luteinizing hormone, which is made from the urine of a pregnant woman, and acts upon the ovaries. We have not treated and operated upon enough cases to say how it would affect the ovaries of the patients if used long enough. I believe that it would be possible to produce some such cystic mass as this tumor in the ovary if we used it long enough. Women with chorio-adenomas and hydatidiform mole have five or ten times as much luteinizing hormone in the urine as does the normal woman. It seems to me that the urine of a case similar to Dr. Gustafson's, or any chorioepithelioma or hydatidiform mole, should show quantitatively an increase in this hormone, and thus a diagnosis could be made. Whether there is a standard or not I do not know; but Aschheim and Zondek say it is possible to make a diagnosis in that way. I think it is important to have the patient's urine tested again to be sure there is no recurrence of mole or epithelioma in the uterus, because if it does occur it may mean the development of a chorioepithelioma. The literature as far as I can tell states that chorio-adenoma, which is the nonmalignant type of chorioepithelioma, develops following hydatidiform moles, whereas choriocarcinoma, which is very malignant, usually follows normal pregnancies.

We have had about fifteen hydatidiform moles in this hospital and about seven chorioepitheliomas. In only one instance was there

any mention of the ovaries, and in that case there were small cystic ovaries, but no mention was made of enlargement, so I do not believe large cysts such as these are as common as one would be led to believe from various textbooks.

DR. MALLORY: Have you any comment, Dr. Gustafson?

DR. GUSTAFSON: There does not seem to be much more to say. I was very much interested in looking up the series of cases in the Boston Lying-In Hospital during the past eighteen years.

Of course it comes down to the way to treat these cases. In some of the literature, especially in the writings of Dr. Schumann of Philadelphia, there has been a tendency to favor laparotomy in these cases, doing a hysterotomy and shelling out the mole when one can see exactly how the mole looks and the uterine tissue looks.

In the Lying-In Hospital during the past eighteen years we had eighteen cases of mole with one death occurring in a case where there was so much bleeding when the vaginal examination was made that it was decided to go in above and shell out the mole. That operation was successful enough in itself except that the woman developed sepsis and on the thirty-ninth postoperative day developed intestinal obstruction and was operated on again, but died on about the forty-fifth day after the abdominal operation. The other cases were all delivered from below. One of these patients had toxemia with convulsions. She was three months pregnant and had all the typical signs of eclampsia, of which mole has been mentioned as a cause, and toxemia is more likely to develop in cases of hydatidiform mole. Of course that lends some strength to the theory that it may be found in the placenta or in degenerated placenta.

This case I treated very conservatively. I did not want to dilate and curette and invite a lot of bleeding; I wanted to wait until the patient started in labor herself. Two other patients of mine have been treated the same way perfectly satisfactorily. I feel that it should be done very carefully, especially since there is a possibility of invasion of the uterine musculature by the mole. It also seems, however, that the development of the Aschheim-Zondek test will give us evidence of any remaining mole or developing chorioepithelioma by the quantitative pregnancy test which was mentioned by Dr. Meigs. If we can shell the fragments out by hand and as long as the uterus is shutting down and consequently the uterine wall getting thicker, it is safe to curette carefully with a large blunt curet. It seems to me that abdominal hysterotomy should be reserved for the rare cases that fail to respond to treatment below.

So far as the diagnosis is concerned, there is no question that it could have been made much earlier by means of a quantitative Aschheim-Zondek test. Sometimes one feels that it is just as well not to spend an extra ten dollars of a patient's money when the diagnosis is going to come out sooner or later.

DR. MEIGS: I did not know that anyone here could do the quantitative test.

DR. GUSTAFSON: In Philadelphia they do it quite often. What they do is to dilute the urine. It is said that in a case of hydatidiform mole as little as one thirtieth of one cubic centimeter of urine will give a positive reaction.

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## THE LAW PROVIDING FOR THE EXAMINATION OF ALLEGED CRIMINALS BY PSYCHIATRISTS

IN 1921 the Legislature of Massachusetts was led to incorporate in its statutes a law which gives authority to the courts to have those charged with murder, and awaiting trial, examined by two psychiatrists for the purpose of receiving from the examiners a report which may be of assistance to the court in dealing with the defendants.

In the cases against three persons alleged to have committed murder (who will soon be tried before a Judge of the Superior Court of Massachusetts), Dr. L. Vernon Briggs and Dr. Earl K. Holt have been selected to examine these persons.

Dr. Briggs was the advocate of this law, and his name has been associated with it. Dr. Holt is the Superintendent of the Medfield State Hospital for the insane.

A very general approval of this law has been expressed repeatedly and the application of its provisions has, in previous court trials, changed the atmosphere of the courts which have tried murder cases since the passage of the act.

Previous to 1921, the divergent opinions of medical experts in murder trials reflected unfavorably on the medical profession, and led the courts in many instances to pay comparatively scant attention to the testimony of physicians. The reactions of juries were equally unsatisfactory.

In the cases of the accused who will soon be tried, where the opinions of Dr. Briggs and Dr. Holt will be before the court, the lawyers for the defendants have been quoted in the newspapers as planning to attack the present law and to employ experts of their own selection to combat the opinions of the psychiatrists appointed by the court.

Publicity has been given to the alleged fact that one of the defendants was formerly examined by Dr. Abraham Myerson, and that he (Dr. Myerson) has been permitted to be present during examinations conducted by Drs. Briggs and Holt.

Dr. Myerson's standing gives promise of no likelihood of unethical behavior, but if men of less experience participate in the court procedures, the attitude of the courts and the behavior of the doctors will be watched with interest. It is not at all probable that, after these years of dealing with criminals under the present law relating to the psychiatric study of defendants, the present system will be found to be defective; because lawyers of standing have not found reason for attacking it and the feeling seems to exist that justice has been promoted. Massachusetts has, thereby, been accredited by other states as having found a better and more dignified way of using expert testimony than is in vogue elsewhere.

## GENERAL PARESIS AND MALARIA

IN the monthly abstract *Journal of the Public Health Service* for February 1934, there is reference to an article in *Bull. de l'Office internat. d'hyg.* pub., Paris, 1933, XXV, 1769 by A. Lutrario, which seems to indicate that persons who have had malaria are less likely to develop paresis, because in those areas where rates of deaths due to malaria were highest, the rates for general paresis were unusually low. For example, in Liguria the death rate for malaria was only 0.2 per 100,000, whereas the death rate for general paresis was 2.9 per 100,000.

The treatment of patients with general paresis by artificially induced malaria, according to some reports, has resulted in amelioration of the paretic symptoms.

## THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

JOSLIN, ELLIOTT P. B.A., Ph.B., M.A., M.D. Harvard University Medical School 1895. Medical Director, George F. Baker Clinic of the New England Deaconess Hospital. His subject is "Coöperation in the Care of the Patient." Page 615. Address: 81 Bay State Road, Boston, Massachusetts.

CRONE, NEIL LOUIS. M.D. Harvard University Medical School 1931. Former House Officer, Massachusetts General Hospital. His subject is "The Treatment of Acute Poliomyelitis with the Respirator." Page 621. Address: Massachusetts General Hospital, Boston, Massachusetts.

MORRISON, LAWRIE B. M.D. University of Vermont College of Medicine 1902.* The construction of his contribution was in association with

MORRISON, SIDNEY L. M.D. University of Vermont College of Medicine 1910. Roentgenologist, New England Baptist Hospital, Faulkner Hospital, Robert Breck Brigham Hospital, and Corey Hill Hospital. Address: 370 Marlborough Street, Boston, Massachusetts, and

DELANEY, JOSEPH H. B.S. Montana State College 1930. Now Senior student at Harvard University Medical School. Address: Harvard University Medical School, Boston. Their subject is "Herniation of the Fundus of the Stomach Through the Esophageal Hiatus: With Special Reference to its Roentgenologic Diagnosis." Page 624.

RICHARDS, ESTHER LORING. B.A., D.Sc., M.D. Johns Hopkins University School of Medicine 1915. Associate Professor of Psychiatry, Johns Hopkins University. Associate Psychiatrist, Johns Hopkins Hospital. Physician-in-Chief, Phipps Psychiatric Dispensary, Johns Hopkins Hospital. Lecturer in Mental Hygiene, School of Hygiene and Public Health, Johns Hopkins University. Visiting Psychiatrist, Baltimore City Hospitals. Her subject is "Practical Features in the Study and Treatment of Anxiety States." Page 633. Address: Johns Hopkins University, Baltimore, Maryland.

EMERY, EDWARD S., JR. A.B., M.D. Harvard University Medical School 1920. Instructor in Medicine, Harvard Medical School. Associate in Medicine, Peter Bent Brigham Hospital. His subject is "The Treatment of Peptic Ulcer Complicated by Hypersecretion." Page 637. Address: 319 Longwood Avenue, Boston, Massachusetts.

*Deceased.

KASANIN, JACOB. B.S., M.S., M.D. University of Michigan Medical School 1921. Clinical Director, State Hospital for Mental Diseases, Howard, Rhode Island. Formerly Research Associate, Boston Psychopathic Hospital, Boston, Massachusetts, Lecturer in Psychiatry, Massachusetts University Extension and Brown University. His subject is "Leucocytosis in Mental Disease." Page 641. Address: Howard, Rhode Island.

WAITE, FREDERICK C. B.Litt., A.M., Ph.D. Harvard University 1898. Professor of Histology and Embryology, Western Reserve University since 1906. Formerly Assistant in Anatomy, Rush Medical College. His subject is "Dr. Lucinda Susannah (Capen) Hall." Page 644. Address: Western Reserve University, Cleveland, Ohio.

## MISCELLANY

### THE PROMOTION OF DR. BAINBRIDGE

Dr. William Seaman Bainbridge has been promoted to the position of Medical Director in the United States Naval Medical Reserve with the rank of Captain.

### INTERESTING COMPARISONS OF NORTH CAROLINA AND MASSACHUSETTS

According to a weekly newspaper published in Moore County, North Carolina, 371 of the 1833 physicians of that state, or more than one-fifth, did not receive more than $1,000 during the fiscal year 1932-1933.

The annual occupational license fee for doctors in North Carolina is $25.00, and those in the income class of less than $1000 were obliged to pay only one-half of the full license fee, or $12.50.

It was alleged in this report that 309 physicians are practicing in North Carolina, but not paying the required State license tax. The population of North Carolina for 1931 was 3,170,276, and in that year there were 2372 physicians making an average of one doctor to 1336+ people. In that year there was one doctor to only 644+ people in Massachusetts.

Massachusetts doctors do not have to pay an annual occupational tax. Although the average clientele of the North Carolina doctor is more than twice that of his brothers in Massachusetts, we do not hear of our doctors migrating to North Carolina except for recreation, or because of a less severe winter climate.

### VENEREAL DISEASE INFORMATION

For a number of years the U. S. Public Health Service has been publishing, for the information of physicians, health officers, and others, a monthly abstract journal known as *Venereal Disease Information*. This publication usually contains one original

article on a subject of general interest in connection with the venereal diseases and numerous abstracts from the current literature pertaining to these diseases. In the preparation of this abstract journal, more than 350 of the leading medical journals of the world are reviewed and abstracts made of the articles on this subject.

The cost of *Venereal Disease Information* is only fifty cents per annum, payable in advance to the Superintendent of Documents, Government Printing Office, Washington, D. C. It is desired to remind the reader that this nominal charge represents only a very small portion of the total expense of preparation, the journal being a contribution of the Public Health Service in its program with State and local health departments directed against the venereal diseases.

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## A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

### Cancer of the Intestinal Tract*

BY WILLIAM M. SHEDDEN, M.D.

Cancer education is worth while. It is shortening the time between the discovery of the disease and the receiving of proper treatment. It need not frighten the listener, it should bring hope, for there is a stage with every cancer *when it is curable* and it is with the desire to get more curable cases to seek early and adequate treatment that this brief talk is offered. Delay is dangerous.

You have often been told that cancer is like a flaming match in the woods. To extinguish the tiny spark at once is a simple matter but to fight it when it has grown into a large monster and has spread to distant parts; that is quite a different matter.

One of the reasons why so many people die of cancer lies in the fact that the disease usually exists for some time before it is properly treated.

The intestine is really quite a wonderful piece of anatomy. Do you realize that the average adult carries around inside of him a tube twenty-eight feet long, twenty of it small and the remainder large intestine? The small intestine is mostly concerned with digestion of food and the large intestine is concerned with absorption of water. The large intestine is festooned across the abdomen, swinging from two fixed parts, one high up in the left abdomen and the other high up on the right. Low down on the right side in the average normal man or woman the small intestine joins the large and it is here that resides our famous friend, or enemy, the appendix. The rectum which is the last six inches of the large intestine lies low down in the abdomen in the midline.

Connected with this long twenty-eight foot tube are many little blood vessels and also a network of little tubes called lymphatics. We are deeply concerned with these little lymphatic tubes for they and the veins can carry cancer cells and scatter them over the body.

#### DANGER SIGNALS

A point that must be emphasized is that early cancer of the intestine may be present without giving any symptoms whatsoever, for there is a period, sometimes long and sometimes short, when the cancer is not large enough to disturb the intestine. "Well—how—" you may ask, "are we going to be saved from this disease if it gives no symptoms?" Simply by going to your doctor once or twice a year for a routine health examination and this should always include a rectal examination. False modesty should never permit us to neglect that part of the examination.

Let us now consider *early symptoms*. First *any change* in bowel habits no matter how slight should be considered important until proved otherwise. The change may show itself in increasing constipation. Don't treat yourself for this or let the druggist prescribe. Don't take oil or cathartics unless suggested by your doctor. I'll tell you why; early symptoms may disappear under such treatment but the cancer remains. Oil allows material to pass through a channel narrowed by disease whereas if the oil were not taken the discomfort would take the patient to a doctor and thus a diagnosis would be made.

A second early symptom may be a slight attack of diarrhea. Of course this sort of an upset is common, while cancer of the intestine is uncommon. However, more than one such attack within a period of a few days is an indication that the individual should see a doctor. Chronic diarrhea in an elderly person is often due to a malignant growth. But don't forget that cancer is not confined alone to elderly people. I have recently investigated over thirty cancers in boys and girls under twenty. Of course, this is rare, but it does occur.

A third early symptom of intestinal cancer is a persistent mild stomach upset, indigestion, or dyspepsia or discomfort in the pit of the stomach, though usually *early cancer* does not disturb either the appetite or the digestion.

Persistent rumbling of gas may be a warning signal.

Pain is rarely important as an early symptom probably because the nerves running from the intestines are so arranged that the mere presence of a tumor does not cause pain.

A frequent urge to relieve oneself of intestinal contents is frequently an early symptom if the growth is low in the intestine, that is near the outlet.

Blood may of course come from anywhere along the intestine. If it is bright it probably comes from the lower end. If it is black it probably comes from a higher level.

*December 22, 1933. Station WBZ. 4:30 P.M.

### LATE SYMPTOMS

If the individual is unfortunate enough to have neglected the disease either consciously or through ignorance long enough, the tumor occasionally becomes large enough to cause a partial or complete blockage. Then at once the symptoms become more acute and cramping pain appears, usually in the lower abdomen, the abdominal wall becomes blown out with gas, there is vomiting and nausea. It is our hope that the sort of a talk you are hearing now will make the picture I have just painted appear less and less frequently. Other late symptoms are anemia, weakness, shortness of breath, and loss of weight. But I must warn you at this time not to be misled by an appearance of good nourishment and weight or color. The haggard emaciated appearance associated in the minds of many with cancer is an appearance noted in the last stages of the disease and usually when nothing more can be done.

Here is what you have a right to expect from an examining doctor: First, that he is respected by his associates. Secondly, that he will take a careful history of the symptoms. Thirdly, that he will perform a careful examination of the whole body and this examination must always include an examination of the rectum with the gloved finger. The importance of this last cannot be overestimated. No patient should be allowed to assume that his symptoms are coming from piles until cancer has been ruled out.

Above the reach of the finger the intestine can be examined by a simple but ingenious instrument known as a proctoscope. The proctoscope is an electrically lighted tube. This can be introduced from below with only slight discomfort and can be done as an office procedure. If your doctor suspects trouble and is not accustomed to this instrument he will refer you to someone who is. To examine the region above that which can be viewed with the proctoscope we made use of the barium enema. Here the doctor examines the patient under an x-ray and watches the course of a small stream of barium introduced from the lower outlet of the intestine. He notices whether it flows up over any suspicious irregularity or meets any obstruction.

X-rays are notoriously unreliable so far as the rectum goes, but above that point are extremely useful. We sometimes also give a small amount of barium by mouth and carefully watch it with the aid of x-ray as it travels down through the intestine.

### CANCER PREVENTION

Over and over again attempts have been made to connect the causation of cancer with some article of food, whether eaten to excess, insufficiently eaten, or harmful because of some property which it was supposed to possess or be deficient in. But when subjected to the cold analysis of science every theory connecting food with cancer has broken down. No one thinks it is sensible to abuse the intestines, but beyond that we cannot go. It has been stated that hemorrhoids, that is piles, and constipation, are two factors which cause cancer. The best evidence that we have at hand goes to show that this has never been proved. Of course, this does not mean that piles and constipation should be neglected. But it does mean that you should not fear that because you have either piles or constipation you are going to get cancer. Only if your constipation is increasing, should you be disturbed.

### TREATMENT

Pills or other medicines by mouth never have and never will cure cancer of the intestines. Ointments and salves never have and never will cure cancer at or near the lower outlet. If the doctor is experienced in treating this disease he will have at his disposal, surgery, radium, x-ray, and an electric instrument known as diathermy. Intelligent use of these means in an early case will result in a high percentage of cures. Don't let anyone tell you that cancer of the intestines is always incurable. It is many times curable, always if removed early enough. It is not sensible to avoid proper treatment by a competent doctor. It is not sensible to fear treatment.

Delay is dangerous!

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## DE MEDICIS CETERISQUE*

(Meditationes ab aliqua lecto affixa)

De Medico Patre

Hic est vir qui semper dicit
Tempus omnia mala vicit
Sed quod tempus longum est
Ad scient(iam) eundum est.

De Medico Medicamentorum

Hic est vir ingeni fortis
Qui delet terrorem mortis
Cum anemiam suspicat
Aut venenum plumbi captat
Omne gaudium prohibet
Iecur ferrumque praescribit.

De Medico Orthopedico

Hic est vir novioris artis
Doctus posterioris partis
Modis aevi saevi utens
Corpora et bracchia trahens
Costas muris saxi cingit
Caud(am) equinam punctis pungit.

De Medico Nervorum Docto

Hic est vir qui malleum portat
Genus, talos, cutem temptat.

De Medico Morborum Docto

Hic vir semina reperit
Longam vestem albam gerit
Urinam in testis tegit
Sanguinem in urnis cogit

*These verses were written by the sister-in-law of a Boston doctor while she was confined to bed by a long illness. The fact that the *Journal* publishes this poem proves that the Editors have a high opinion of the clinical ability of the *Journal's* readers.

Omnia minima inspectat
Morbus mult(um) cum delectat.

De Medico Radiorum Incognitorum
Hic est vir cui omnes credunt,
Et cui victimae se dedunt,
Haruspex; futurum dicit
Simul viscera inspicit.

De Medico Clinico
Hic dolorem nobis parcit
Omnia post tergum facit
Sacralem hiatum quaerit
Eius hasta alt(e) inhaerit
Monstrum dirum ac horrendum
Corpus reddit moribundum.

De Medicis Domi
Iuvenes albo vestiti
Vos scientiae periti
Quantum eheu cognoscetis
Cum in mundum venietis!

De Nutricibus
Puellae nondum maturae
Quales feminae futurae?
Multum doctae, semper bonae,
Sed sub tegmentis personae.

De Fluoroscopia
Lux fiat! nunc omnes agunt
Fluctus barii insurgunt
Clare per vitrum apparent
Omnia quae celari debent.

De Ventris Exploratione
Facilis descensus est
Averno. Non ventris est.
Sed mihi nunc est probandum
Cur utramque descendendum.

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## FALLACIES ABOUT HOME TREATMENT OF THE EYES

Some popular fallacies about home treatment of the eyes are described by Dr. William L. Benedict of the Mayo Clinic in the winter issue of *The Sight-Saving Review*. He says:

"There is very little understanding on the part of the public as a whole that a most potent antiseptic agent is supplied to the eye by natural means: that is, by the tears. Laboratory experiments have shown that for most disease-producing organisms, normal tears are hundreds of times more effective in protecting the eye than solutions of drugs in such strength as can be borne. The tears are supplied in amounts that are properly regulated, and counteract the effect of most air-borne bacteria.

"Because the general public has learned that many eye lotions are put up in a solution of boric acid, the better informed immediately turn to this solution as the one safe substance that can be used for any disorder of the eye. Hence, we find that many people are using an eye cup, giving the eye a daily bath in boric acid solution, in the hope that their disorders will soon pass away and the necessity for visiting a physician may be obviated.

"The healing properties of boric acid are infinitesimal. It is used chiefly by physicians as a vehicle to carry small dilutions of more potent drugs; and because it retards the growth of fungi or of contaminating bacteria, it forms a convenient way to dispense medicines for the eye. . . . The practice of giving eye baths with any solution when eyelids are not diseased, except on the advice of an oculist, should be discouraged. . . .

"There are very few remedies for the eyes that are specific. There is not one bit of scientific proof that there is any drug or other agent by which the development of cataract can be generally retarded, or by which cataracts can be removed without operation. A great deal of experimental work is being carried out in laboratories and clinics, by members of the medical profession, with the hope that some means will be found to obviate the necessity of operating for cataracts.

"I think it is well known and appreciated by people at large that there are no miraculous cures for any diseases or disorders of the eye. The faith that our grandmothers had in the use of goat's milk, sauerkraut juice, snake oil, milk and honey, or tobacco juice, has been largely dissipated by an intelligent public. The price the public has had to pay for its experience has been the loss of sight for thousands of people through misinformation, through misdirection, through misplaced confidence, and through ignorance.

"People at large now know the value of cleanliness and the necessity of seeking medical advice when the symptoms of disease appear. They have learned that it is not advisable to attempt to remove foreign bodies from the eye in workshops where the instruments used may be so contaminated as to result in disease of the eye more serious than would be caused by the original injury. They have learned the value of prophylactic treatment of the eyes of the newborn, which has reduced the incidence of babies' sore eyes and consequent blindness to a great extent.

"When we consider the great variety of hazards to which our eyes are exposed in the ordinary pursuits of life, the wonder is that many more people are not deprived of the faculty of vision. Serious injuries are relatively infrequent as compared with injuries of other parts of the body. This is partly because the eyes are well protected by being situated in a bony orbit, and partly because they lie behind the eyelids, which can be quickly closed.

"The incidence of diseases of the eyes caused by infection has been greatly reduced by measures of personal hygiene which people in general have adopted, and because they have come to understand that diseases such as granulated lids and other acute inflammation can be spread by personal contact. There has been an effective campaign also against

industrial injuries to the eyes. Blindness has been materially reduced as a result of campaigns carried out by members of the medical profession and by organization of the laity. There is yet great work to be done in educating the public in the care of eyes that have been injured, in the treatment of minor ailments or diseases of the eyes, and in the treatment of diseases of the eyes in their early stages."

## RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR FEBRUARY, 1934

Measles is unusually prevalent in the eastern part of the state. Present indications point to a year of increased incidence as in 1928.

Diphtheria reached its lowest reported incidence for Massachusetts, regardless of month or season.

Lobar pneumonia reports show nothing remarkable.

Scarlet fever is running somewhat lower than usual.

Typhoid fever, for the first two months of the year, compares favorably with last year's record figures.

Pulmonary tuberculosis is at its lowest reported February incidence.

Whooping cough shows a somewhat increased prevalence.

Anterior poliomyelitis, chicken pox, epidemic cerebrospinal meningitis, German measles, mumps, and tuberculosis other forms show nothing remarkable.

### RARE DISEASES

*Anthrax* was reported from Haverhill, 1; Lynn, 1; total, 2.

*Dysentery* (*Amebic*) was reported from Boston, 2; Canton, 1; Clinton, 1; total, 4.

*Dysentery* (*Bacillary*) was reported from Boston, 1; Medfield, 1; total, 2.

*Encephalitis Lethargica* was reported from Boston, 1; Holyoke, 1; Westfield, 1; total, 3.

*Malaria* was reported from Chelsea, 1.

*Cerebrospinal Meningitis* was reported from Boston, 1; Dalton, 1; Fitchburg, 1; Grafton, 1; Lowell, 1; Peabody, 1; total, 6.

*Pellagra* was reported from Revere, 1.

*Septic Sore Throat* was reported from Beverly, 3; Boston, 6; Braintree, 1; Everett, 1; Medford, 2; Milton, 1; Stoneham, 1; Taunton, 3; Upton, 1; Weston, 1; Worcester, 1; total, 21.

*Trachoma* was reported from Boston, 1.

*Trichinosis* was reported from Boston, 2; Lexington, 2; total, 4.

## MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

MONTHLY REPORT FOR FEBRUARY, 1934

| Disease | Feb. 1934 | Feb. 1933 | Average* |
|---|---|---|---|
| Anterior Poliomyelitis | — | 1 | 3 |
| Chicken Pox | 1157 | 1106 | 1005 |
| Diphtheria | 27 | 91 | 242 |
| Dog Bite | 356 | 318 | 277 |
| Epidemic Cerebrospinal Meningitis | 6 | 1 | 9 |
| German Measles | 57 | 51 | 128 |
| Gonorrhea | 416 | 427 | 441 |
| Lobar Pneumonia | 552 | 466 | 653 |
| Measles | 8637 | 982 | 1654 |
| Mumps | 488 | 718 | 729 |
| Scarlet Fever | 972 | 1499 | 1462 |
| Syphilis | 350 | 340 | 287 |
| Tuberculosis Pulmonary | 205 | 213 | 330 |
| Tuberculosis Other Forms | 33 | 35 | 39 |
| Typhoid Fever | 9 | 4 | 11 |
| Undulant Fever | — | — | — |
| Whooping Cough | 1273 | 732 | 799 |

*Average number of cases for February during the preceding five years.

# CORRESPONDENCE

## THE POLICE UNIFICATION BILL

The Massachusetts Medical Society
Office of the President, Dr. William H. Robey
202 Commonwealth Avenue
Boston, Mass.

March 13, 1934.

The *Editor*,
*New England Journal of Medicine*,

Since the support of any measure by the Massachusetts Medical Society cannot be given without the consent of the Council, I am asking you to print the following letter in order that the members of our Society may take individual action. The letter explains its importance and we hope that members of the Massachusetts Medical Society throughout the State will read it with care.

Very truly yours,
WILLIAM H. ROBEY, M.D.

Committee For Public Safety
11 Beacon Street
Boston, Massachusetts

March 9, 1934.

Dr. William H. Robey,
Boston, Mass.
*Dear Sir:*

We ask that your organization support in writing, and by the presence of its officers at hearings at the State House, the Police Unification Bill of the Committee for Public Safety.

We also ask that you obtain in writing from your

members, to such an extent as is possible, the backing of this Bill. Our reason for this request is as follows:

1. Only by such a Bill can the 4,600,000 citizens of the State be protected.
2. The police departments of Massachusetts are in no manner affected by the control of the Commissioner of Public Safety and his four Associate Commissioners, except when, as, and if members of the local departments cease to be efficient and honest police officers.
3. The morale of such a coördinated police force throughout the State will form the fourteen to sixteen thousand police officers into a group respected by the citizens and feared by the lawbreakers.
4. This unified police body will depend no more on political favor or political appointment.
5. Police officers who "go wrong" under such a system will be tried by their own Boards in their own local departments, and the findings of these Boards will be subject to appeal to the Disciplinary Board at the head.
6. Accoutrement of every nature, including modern crime-fighting apparatus, will be at the disposal of the local departments.
7. Training school, civil service certification, and method of advancement are things the whole State is in agreement on.

Last but not least, the gradually mounting opinion of this State, expressed by telephone, letter and telegraph, and by personal visits to our offices, convinces us that the mass of citizens of this State are in accord for this Bill. The press is unified and definitely for it. The legislative body has definitely turned to its support.

We need your backing and it is your duty to assist in giving your State a real splendid force that will serve as a model to all other states. The heads of families will appreciate the dangers they are in under the present system and will really get to work for this Bill.

Very truly yours,

COMMITTEE FOR PUBLIC SAFETY,
By ROGER W. CUTLER,
*Secretary-Treasurer.*

## RECENT DEATH

**DORCEY**—JAMES EDMUND DORCEY, M.D., aged 80 years, of 174 Harrison Avenue, Boston, died at the Massachusetts General Hospital, March 13, 1934.

He was graduated from the Harvard Medical School in 1878, and after serving in charge of the Albany Hospital for a time, he returned to Boston and opened his office, devoting himself to family practice.

Dr. Dorcey was known as a dispenser of charity on a large scale. He joined the Massachusetts Medical Society in 1880, and retired in 1929.

He was a member of the Charitable Irish Society, the Foresters, and several other organizations. His wife died three years ago. He is survived by his daughter, Miss Elizabeth Dorcey.

## NOTICES

### ANNOUNCEMENTS

PERCY A. BROOKE, M.D., announces the opening of an office at 27 Elm Street, Worcester, Mass., Rooms 206-207.

DR. RICHARD CHUTE, who has been associated for a number of years with his father, the late Dr. Arthur L. Chute, announces that he will continue the practice at the same office, 352 Marlborough Street, Boston. Telephone: Kenmore 9710.

ROSS MCPHERSON, M.D., and ROGER H. DENNETT, M.D., will remove their office to 842 Park Avenue, corner of 76th Street, New York City, on April 1, 1934.

### RADIO HEALTH MESSAGES

MARCH, 1934

**Sponsorship: Public Education Committee of the Massachusetts Medical Society and Massachusetts Department of Public Health.**

**Courtesy WBZ. Fridays, 4:30 P.M.**

March

**23 How to Keep the Well Child Well**

**30 Résumé of the Year's Work**

HEALTH QUESTION BOX

**Sponsored by Massachusetts Department of Public Health. Fridays, 4:40 P.M.**

RADIO HEALTH FORUM

**Queries from the public are answered under the sponsorship of the Department of Public Health.**

**Courtesy WEEI. Fridays, 5:00 P.M.**

**Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.**

SPECIAL

**Courtesy WEEI. Fridays, 1:15 P.M.**

**Glimpses into the History of Public Health in Massachusetts together with the Functions and Activities of the Massachusetts Department of Public Health, Blended with Classical Music.**

# REPORT AND NOTICES OF MEETINGS

## FAULKNER HOSPITAL CLINICAL MEETINGS

On Thursday afternoon, March 1, at 5:00 P.M., the regular monthly meeting of the Staff of the Faulkner Hospital was held at the hospital.

Two of the cases which had come to autopsy during the month were presented for discussion.

The first one emphasized the danger which is always present in a person who has gallstones. This patient had suddenly been taken ill with abdominal pain and vomiting. This was associated with the appearance of jaundice and also with a very small output of urine as the disease progressed. By the time the patient arrived in the hospital she was in such a condition that operation was not considered advisable, and before sufficient improvement developed so that an operation could be attempted, she died. At the postmortem examination there was found acute infection in the gallbladder associated with gallstones, an acute pancreatitis and an acute nephritis.

The other case was especially instructive as it supports the contention of pharmacologists that avertin may be exceedingly dangerous. This was the case of an elderly man with Paget's disease and a fracture of the femur. Avertin was given on two occasions three days apart; in the first instance in an attempt to reduce the fracture by the closed method; in the second, when the leg was operated on in order to reduce the fracture by the open method. Following the operation the patient's temperature became elevated although the pulse rate did not. This elevation of temperature persisted. Four days after the second dose of avertin, jaundice developed. The jaundice became more intense but apparently was not due to obstruction, because bile was always present in the stools. A day or so later the urine became scanty and the patient became drowsy. Death occurred ten days after the second dose of avertin and at postmortem examination there was found an extensive, acute, degenerative lesion in the liver and an acute degenerative lesion in the kidney, both of which were considered to be toxic in origin. There was a bronchopneumonia and a streptococcus septicemia, both of which were thought to be terminal and not the cause of the degenerative lesions in the liver and kidney. These were supposed to be due to avertin.

Dr. Burton E. Hamilton then gave a short talk on "The Recent Advances in the Clinical Use of the Electrocardiogram" in which he briefly outlined the history of clinical electrocardiography. Following this he demonstrated lantern slides of electrocardiograms illustrating the common disorders of the heart beat and myocardial changes. The object of this demonstration was to enlighten the members of the staff concerning the significance of routine electrocardiograms when taken and interpreted at their request in the Faulkner Hospital, and to suggest the place that electrocardiography may be expected to take in clinical diagnosis. Dr. Hamilton's observation suggested that only in rare cases can the electrocardiograms be expected to furnish us a valuable diagnosis otherwise not obtainable, but that it has become a definite, though perhaps slight aid, in the routine diagnosis and control of the majority of cardiac patients to those comparatively few physicians who have learned to read and evaluate it. Dr. Hamilton has found that the majority of physicians ordering electrocardiograms learn very little from the tracings themselves, and more often than not, gain very little information from the routine interpretation of the electrocardiograms. What the average request for an electrocardiogram usually amounts to is a direct request for advice, such as the implied questions — "Having seen the electrocardiogram do you think the patient has angina, a coronary occlusion, or is there anything wrong with the patient's heart or is the patient simply neurotic?" These and other questions are intensely appropriate. The desirability of accurate answers is evident. Though in many cases the answer cannot be given, in others it can, and in still more cases useful suggestions for methods of settling the question can be offered.

In order to enhance the value of the reports of electrocardiograms in this hospital, it has been suggested that the physician requesting the report give as clear an idea of the situation as possible. For instance, he should among other points state the age of the patient, whether digitalis has been given and if so how much, and the reason for requesting the electrocardiogram; to settle the nature of arrhythmia; to determine whether the heart may be the cause of a patient's fainting attacks and so forth.

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The next meeting will be held at the Faulkner Hospital at 5:00 P.M. on Thursday, April 5. In addition to the usual clinical pathological conference on the cases which have come to autopsy during the month Dr. Edward L. Young, Jr., will give a short talk on "Some of the Difficulties of Gallbladder Diagnosis." All physicians who are interested are cordially invited.

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## CARNEY HOSPITAL CLINICAL MEETING

The Staff of the Carney Hospital will conduct clinical meetings on the last Wednesday of each month (excluding June, July, August, and September). The next clinic will be held at the hospital on Wednesday morning, March 28, between the hours of 9:30 and 12 o'clock.

All physicians are cordially invited to attend these clinics.

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## THE NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the Society will be held in the Faulkner Hospital, March 27, 1934, at 7:15

P.M. Telephone Jamaica 2740. *Please note change in hour of meeting.*

Business: Action on the minority report presented by Dr. Henry M. Landesman.

Communications: 8:15 P.M. Symposium on Varicose Veins. Dr. Henry H. Faxon, Dr. Edward A. Edwards.

Discussion opened by Dr. E. E. O'Neil.

Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*
1695 Beacon Street, Brookline.

## THIRTIETH ANNUAL MEETING OF THE NATIONAL TUBERCULOSIS ASSOCIATION

This meeting will be held at the Netherland Plaza Hotel, Cincinnati, Ohio, May 14, 15, 16, 17, 1934.

Opening General Meeting, Monday, May 14, 8:15 P.M.

Address of the President, Dr. Stuart Pritchard, Battle Creek, Michigan.

Report of the Managing Director, Dr. Kendall Emerson, New York.

Award of the Trudeau Medal, Dr. Esmond R. Long, Philadelphia, Pa.

Report of the Committee on Nominations, Dr. James J. Waring, Denver, Colorado.

Address—"The Rôle of Tuberculin in Tuberculosis Control," Dr. Marion Dorset, Washington, D. C.

Showing of Motion Pictures on Growth of Acid-Fast Bacilli, Ralph W. G. Wyckoff, Ph.D., New York.

For details of the program apply to The National Tuberculosis Association, 450 Seventh Avenue, New York City.

## THE AMERICAN ASSOCIATION ON MENTAL DEFICIENCY

The annual meeting of the American Association on Mental Deficiency will be held at the Hotel Waldorf Astoria, New York, May 26, 27, 28 and 29, 1934. The Saturday session, May 26, will be given over to the sociological, psychological and the special educational aspects of the problem in order that social workers and school teachers may have an opportunity to attend without interfering with their regular duties. The Tuesday afternoon session will be a conjoint meeting with the American Psychiatric Association. Data as to the program may be obtained from the Secretary, Dr. Groves B. Smith, Godfrey, Illinois.

## HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, March 27, at 8:15 P.M.

PROGRAM

Presentation of Cases.

Studies with Diuretic Drugs. By Drs. Marshall, N. Fulton, William Evans, Jr., A. H. Bryan and E. A. Stead, Jr.

Studies on Sulphur and Protein Metabolism in Bright's Disease. By Drs. G. P. Grabfield and B. Prescott.

JOHN HOMANS, M.D., *Secretary.*

## SUFFOLK DISTRICT MEDICAL SOCIETY

The March meeting of the Suffolk District Medical Society will be held on Wednesday, March 28, 1934, at 8:15 P.M., in the Evans Auditorium at the Massachusetts Memorial Hospitals.

PROGRAM

"Gall-Bladder Pathology in Children." Clifford D. Harvey, M.D.

"Ocular Pathology in the Newborn." William D. Rowland, M.D.

"Streptococcus Viridans Mastoiditis and Meningitis with Recovery." Leighton F. Johnson, M.D.

"X-Ray Diagnosis of Cardiac Pathology with Demonstration of the Mechanical Heart." George Levene, M.D.

"Electrical Oscillations and Their Significance in Electrocardiography." Samuel H. Caldwell, Sc.D., Massachusetts Institute of Technology.

Discussion by Drs. Lester R. Whitaker, Ralph Hopkins, Maurice Fremont-Smith, William D. Reid.

Refreshments will be served at 10 P.M.

JAMES H. MEANS, M.D., *Vice-President,*
GEORGE P. REYNOLDS, M.D., *Secretary.*

## NEW ENGLAND DERMATOLOGICAL SOCIETY

The Annual Meeting of the New England Dermatological Society will be held at the Boston City Hospital on Wednesday, April 11, at 3:00 P.M.

J. HARPER BLAISDELL, M.D., *Secretary.*

## SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

**March 23**—New England Pediatric Society will meet in Boston. 4:15 P. M.—Clinical Program, Children's Hospital, Longwood Avenue, Boston. 6:45 P.M.—Dinner, Vanderbilt Hall, Harvard Medical School. 8:15 P.M.—Evening Program—Boston Medical Library, 8 Fenway.

**March 26**—New England Heart Association will meet in the Evans Auditorium of the Massachusetts Memorial Hospitals, Boston, at 8:15 P.M.

**March 27**—Harvard Medical Society. See notice elsewhere on this page.

**March 28**—Carney Hospital Clinical Meeting. See page 662.

**April 5**—Faulkner Hospital Clinical Meeting. See page 662.

**April 11**—New England Dermatological Society. See notice above.

**April 13, 20 and 27**—Salmon Memorial Lectures. See page 443, issue of February 22.

**April 16**—Boston University School of Medicine to Conduct a Clinical Meeting at Boston City Hospital.

**April 16-20**—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

**April 30**—The American Board of Dermatology and Syphilology. Examinations for Certificates. Address: Dr. C. Guy Lane, 416 Marlboro Street, Boston, for details.

**May 14, 15, 16, and 17**—Thirtieth Annual Meeting of the National Tuberculosis Association. See page 663.

**May 26, 27, 28, and 29**—The American Association on Mental Deficiency. See page 663.

**July 24-31**—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schniz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

**September 3-6**—American Public Health Association, at Pasadena, California. Dr. J. D. Dunshee, Chairman, Local Committee on Arrangements.

**September 4, 5, 6**—International Union Against Tuberculosis will be held in Warsaw. For particulars address The National Tuberculosis Association, 450 Seventh Avenue, New York, N. Y.

## DISTRICT MEDICAL SOCIETIES

### ESSEX SOUTH DISTRICT MEDICAL SOCIETY

**Wednesday, April 4**—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speakers: Dr. Elliott P. Joslin and Dr. Howard F. Root, Boston. Subject: "Tuberculosis Complicating Diabetes."

**Thursday, May 3**—Censors' Meeting, at Salem Hospital, 3:30 P.M.

**Tuesday, May 8**—Annual Meeting. Salem Country Club, Forrest Street, Peabody. Dinner at 7. Speaker to be announced. Subject to be announced.

RALPH E. STONE, M.D., Secretary.
221 Cabot Street, Beverly, Mass.

### FRANKLIN DISTRICT MEDICAL SOCIETY

The next meeting will be held on the second Tuesday of May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland, Mass.

### MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The next meeting will take place in May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, M.D., Secretary.
76 Church Street, Winchester, Mass.

### MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meeting will be held on April 25.

T. A. STAMAS, M.D., Secretary.
226 Central Street, Lowell, Mass.

### NORFOLK DISTRICT MEDICAL SOCIETY

**March 27**—See page 662.

**April 17**—Hotel Kenmore, 8:30 P.M. Special Business Meeting.

**May**—Annual Meeting. Time, place and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline, Mass.

### NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

**April 5**—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

**May 3**—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.
Norfolk County Hospital, South Braintree, Mass.

### SUFFOLK DISTRICT MEDICAL SOCIETY

**March 28**—See page 663.

**April 25**—Annual Meeting at the Boston Medical Library. Election of Officers. Scientific Program, titles and speakers to be announced.

The Medical Profession is cordially invited to attend these meetings.

JAMES H. MEANS, M.D., Vice-President.
GEORGE P. REYNOLDS, M.D., Secretary,
311 Beacon Street, Boston, Mass.

### WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:

**April 11**—Open date.

**May 9**—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester, Mass.

## BOOK REVIEWS

*The Treatment of Rheumatism in General Practice.* By W. S. C. COPEMAN. Published by William Wood and Company, Baltimore, London. 209 Pages. Price $3.25.

There is nothing particularly new in this book, but there is a good deal of misleading information. For example, the author's differentiation of acute muscular rheumatism, fibrositis and lumbago is not clear. There appears, also, to be very little recognition of the so-called mechanical irritant, producing a short period of joint pain, stiffness or swelling in a hypertrophic arthritic. Again, a number of so-called cured arthritics are mentioned. From the description of these cases, and the short periods they were under observation in an out-patient department, they appear more likely to have been arrested cases of non-articular joint involvement than true *rheumatoid* or *atrophic* arthritis. No mention is made of the length of time one patient, or a group of patients, was followed. A short period of observation obviously gives a different picture than a period covering years' observation of the same patient.

Osteoarthritis, he considers correctly, simply as a manifestation of wear and tear on tissue, which probably would have remained under the same heading in the United States if specialists had not made a distinct disease of it, with all the beneficial as well as objectionable features attendant when one specializes in something that may be debatable.

Treatment at the present stage of medical knowledge is very well presented in this book, but here again one is impressed with the numerous forms and types of treatment suggested. This leads the reviewer more and more toward the point of individualization in treatment; and the general care and treatment of a patient indirectly help the mechanical impairment of joints up to a certain point.

On the whole, the book is a mixture of good and bad. The last chapter, Chapter 23, prognosis and end results, as the author states, is the most important but one which most writers avoid. One's reaction after reading this chapter is that perhaps this might have been a good rule to have followed. However it is a fair summary of one man's observations concerning the prognosis in arthritis, though such optimistic prognosis may well arouse expectations and hopes which may not be fulfilled. On page 207, under the paragraph heading of "Infective and Non-Infective Rheumatoid Arthritis," the author states that if a patient is "seen within two months of its onset it (arthritis) may be cured: within 6 months a cure is still possible; within a year a fairly good result may be anticipated but will take perhaps 6 months to accomplish," that, generally speaking, every additional month the disease progresses more time will be needed for treatment. This statement is followed by a paragraph stating that this disease often be-

comes quiescent in two years and at this stage the mechanical or operative aspect of arthritis is introduced. This consists of mechanical interference, altering anatomical defects, but in no way influences the disease itself. Such an unqualified statement of favorable end-results in the treatment of arthritis, at least of the true rheumatoid progressive type which we see in the eastern part of the United States, seems open to question to say the least. It appears that the writer's conception of the life course of an individual suffering from rheumatoid arthritis is based not on the actual life course of a single individual but rather on a theoretical course made up of segments from the life courses of many individuals.

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*The Enlarged Prostate and Prostatic Obstruction.* By KENNETH M. WALKER, F.R.C.S., M.A., M.B., B.C. Second Edition. Published by Oxford University Press, London. Humphrey Milford, 1933. 223 pages.

Ten years have elapsed since the publication of the first edition of Kenneth Walker's monograph. During this decade, the operation of prostatectomy has been subjected to close scrutiny because of the introduction of new transurethral methods of removing the obstruction at the bladder neck. In the second edition of his book, Walker gives considerable space to the discussion of prostatic resection; his conclusions agree with those of the more conservative urologists in this country.

As a whole, his book is an excellent one, well balanced, sane and practical. Especially good are the opening chapters on the anatomy, physiology and pathology of the prostate gland. His condemnation of the practice of closing the bladder after prostatectomy is in accord with the reviewer's opinion of this illogical method. One may well object to his custom of referring to ordinary prostatectomy as a "total removal" of the gland. It is definitely not this, as both the capsule, which consists of compressed glandular tissue, and the posterior lobe are, or should be, not removed by either the suprapubic or the perineal operation.

Several other isolated statements may also be questioned. For example, Walker says that during prostatic resection the current of fluid should be checked for fear it will wash the fragments into the bladder. Demonstrators of the resection apparatus insist that the current should flow freely at this time, to avoid burning out the electrode. Furthermore, a better view of the region under attack is obtained by free irrigation.

On page 94 the author refers to the risk that hypertrophied prostates may become malignant, and says that this risk has been assessed at 13 to 15 per cent. We doubt the truth of this, although we admit that carcinoma and hypertrophy may develop independently in the same gland. Only occasionally, we believe, does cancer develop in the hypertrophied portions of an enlarged prostate.

In his discussion of methods of anesthesia, Walker, quoting Barney and Shedden, states that "in some 10% of cases in which spinal anesthesia is used a spinal reaction occurs. This reaction is always to be feared and is not infrequently fatal." This observation, which appeared in a paper published in 1923, before the use of ephedrin to support the blood pressure became at all general, would hardly be true of spinal anesthesia as it is employed today.

Aside from these few minor criticisms, the reviewer finds Walker's book to be an admirable example of the surgical monograph, especially valuable for the student and the general practitioner.

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*Surgical Anatomy.* By GRANT MASSIE. Published by Lea & Febiger, Philadelphia. 458 Pages. Price $6.00.

This English manual of Surgical Anatomy, first published in 1930 and favorably reviewed in the *Journal* at that time, aimed to fill a definite place in the teaching of Applied Anatomy from the clinical standpoint. It is extremely difficult for any work of this sort to surpass the classical standard of Treves' Manual; but in its illustrations and in its more modern point of view, this work made an exceedingly favorable impression. In this second edition the entire text has been carefully revised and numerous alterations and additions have been made in the light of current surgical practice. Chief among the additions are new sections dealing with injuries of the Carpus, infections of the Hand, surgical approaches to certain of the Long Bones, and to autonomic nerve system. Some new illustrations have been added bringing the total to 147, of which many are in color and all are admirable. Though not possessed of the amazingly compact inclusiveness of Treves' work, this volume, for the reason stated, remains a very valuable addition to the equipment of the student or teacher of Applied Anatomy.

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*Bone Growth in Health and Disease.* The Biological Principles Underlying the Clinical, Radiological, and Histological Diagnosis of Perversions of Growth and Disease in the Skeleton. By H. A. HARRIS, D.SC.LOND., M.B., B.S. (Lond.), B.Sc. (WALES), M.R.C.S., M.R.C.P., Professor of Clinical Anatomy, University College and University College Hospital, London, Etc. Oxford University Press. London: Humphrey Milford. 1933. 248 Pages.

Your reviewer does not remember having read a more thought-provoking medical book. One of its many virtues is the fact that it suggests and proposes and does not dogmatize or conclude. All its pages are filled with information based on long experience, intensive study and a breadth of knowledge made applicable by an open mind and the priceless faculty of imagination. The thirty-one short, beautifully illustrated chapters are grouped under three main headings. Part I interprets the meaning and

mechanism of the "Lines of Arrested Growth" seen so often in radiographs of the long bones of childhood; Part II discusses bone and cartilage growth with special reference to deficiency diseases; Part III considers the significance of the fundamental processes of growth and repair in skeletal disease.

One may even catch a glimpse of the author's personality from a chance sentence. He is discussing certain starvation experiments on young (and evidently appealing) dogs, and remarks "For obvious reasons, *largely sentimental*, these experiments were not carried over a long period of time."

In Part I, Harris has shown that "During the whole period of growth any severe infection or metabolic disease such as diabetes, induces a premature senescence in growing tissues, and the fact is unfailingly and accurately registered in the structure of the bone by the production of a transverse line of arrested growth."

In Part II will be found interesting conceptions of the growth of bone and an original hypothesis concerning the method of formation of the multinuclear giant cells usually called osteoclasts. He believes that synovial fluid is produced from the detritus of the decaying cartilage cells and matrix resulting from wear and tear at the joint surface. Perhaps this statement needs further explanation, for, while the theory has been previously advanced, it would not seem to be defended by recent investigations.

The author trenchantly criticizes "Wolff's law" and considers that Jansen "has demolished many of the most cherished illustrative examples brought forward in support of the orthographic trajectories demanded by the conception of Meyer-Wolff." He also believes that Dr. Honor Fell's experiments have irrefutably shown that the pattern of the trabeculae in bone is not determined by the Meyer-Wolff law.

A needed word of caution in relation to the application of the results of animal experimentation to human beings appears, in connection with dietary measures, and he pleads for a complete pathological examination as "a check on clinical exactitude".

In Part III there are most interesting and perhaps conception-changing chapters on Achondroplasia, and age changes in the skull, on Osteitis Deformans (Paget's Disease) and on "Glycogen and Phosphatase in the Metabolism of Cartilage". The author considers it possible "that the various types of hyperplasia and degeneration on the one hand and the various perversions of growth, ranging from generalized skeletal disease to localized tumors on the other, are expressions of fundamental nutritional differences".

How extremely suggestive is this little volume of some two hundred and thirty pages may be gathered by quoting the last paragraph of text. "It is evident that the new outlook in biochemistry will necessitate a complete revision of the normal and pathological histology of cartilage and bone, in terms of glycogen, phosphatase, and other substances whose rôle is as yet unknown. Although the implications of this chemical approach to the diseases of bone, joints, and teeth have only recently emerged, it is gratifying that a combined radiographic and histological study of lines of arrested growth should lead to a clearer conception of biological processes and open out a new field which calls for fresh methods of attack in order to elucidate the problems of bone growth in health and disease."

The book will be found to warrant a reading and a re-reading not only by research workers but by all physicians seeking information which will aid them in forming conceptions as to the nature and significance of the basic processes concerned in "Bone Growth in Health and Disease".

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*The Story of Childbirth.* By PALMER FINDLEY. Published by Doubleday, Doran & Co., Inc. 361 Pages. Price $3.00.

This is a unique book, by one of the most forthright and forward looking obstetricians in America, dealing in detail with the accumulated traditions of childbirth, from mythological times. It takes up the taboos and customs of primitive races in relation to procreation, and shows the influence of these on present-day superstitions and misconceptions regarding pregnancy. Particularly, does it stress the complete absence of scientific support of the commonly accepted belief in maternal impressions and their influence in marking a child. It traces the dawn of scientific obstetrical achievement, the development of operative obstetrics, the discovery of the cause of puerperal fever, the discovery of anesthesia and its application to obstetrics. It then discusses modern obstetrical problems, the question of midwives, the question of the still too high maternal mortality rate, the value of prenatal and postnatal care, the question of birth control, child spacing and abortion, the relation of venereal diseases to pregnancy. It closes with an excellent chapter on the "Modern Maternity Hospital."

The book is attractively bound, is easy to read and is well and generously illustrated. It is equally interesting to the medical student, to the doctor, to the lay man or woman. One feels that it should not be allowed to fall into the hands of a pregnant woman. In the opinion of the reviewer, the chief value of the book is that from an authoritative source the layman may learn in a non-technical and interesting manner the facts connected with childbirth, and so may be educated to an understanding of the real need of first-rate obstetrics, and may come to feel an appreciation of the expenditure of time, judgment and effort spent in conducting a woman successfully through pregnancy, labor and the puerperium. It supplies the medical student and the general practitioner with a valuable background of obstetrics past and present on which to build his practical work.

To do these things as well as is accomplished in this book is an achievement.

*The History and Epidemiology of Syphilis.* By WILLIAM ALLEN PUSEY, A.M., M.D., LL.D. Published by Charles C. Thomas, 1933. 113 Pages. 73 Illustrations. Price $2.00.

This book comprises three chapters, each a lecture delivered under the Adolph Gehrmann Endowment at the University of Illinois, College of Medicine, Chicago. It is an elaboration of certain chapters of the author's Syphilis as a Modern Problem, published in 1915. He has added the progress of the past eighteen years in our knowledge of the history of syphilis and our epidemiological experience of the Great War.

The first chapter is the story of the beginning and spread of syphilis over the world to the closing of the fifteenth and the opening of the sixteenth centuries. In emphasizing the theory of the American origin, he has used very carefully the early writings in Spain and in other European countries, as well as Williams' exhaustive studies on prehistoric American bones. The second chapter develops the story of the growth of our knowledge of syphilis during the last four hundred years. It is freely illustrated and gives brief sketches of the important investigators of this period, with hints as to the background of their times. The third chapter is an orderly discussion of the epidemiology treated under (1) the reservoir; (2) the infecting organism; (3) the susceptible host; (4) the means of transmission.

The book is charming in its artistry and physical qualities and in content is what one would expect from the pen of William Allen Pusey who has been responsible for a textbook on dermatology and several works of literary and scholarly value.

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*Peripheral Nerve Injuries.* By LEWIS J. POLLOCK and LOYAL DAVIS. Hoeber's Surgical Monograph. Paul B. Hoeber, Inc., New York, 1933. Price $10.00.

The publication of this monograph is well timed for the profession stood in need of an authoritative exposition of the whole subject of peripheral nerve injuries. Sufficient time has elapsed since the war to permit of proper evaluation of the work done during those days when nerve injuries flowed into the military hospitals of the world in a never-ending stream. The various books that came out and articles published in the current literature could not give us full perspective. This work does give us just that perspective as seen through the eyes of two keen observers, each a man of wide experience in his chosen field.

The book is well written, well illustrated and well printed. The examination of the patient both before and after treatment, anatomy, diagnosis and treatment are all well covered and the bibliography and indexing are well done.

Such a book as this will be of great interest to those who are particularly in this type of work. It will also be a real help as a book of reference to the industrial surgeons and those practitioners who meet an occasional case of nerve injury in general practice.

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*Sex Habits. A Vital Factor in Well-Being.* By A. BUSCHKE and F. JACOBSOHN. Published by Emerson Books, Inc., New York. 204 Pages. Price, $2.50.

Too many of the books on sex written for the lay reader are either superficial or confused by detail; they do not go deeply enough to satisfy the intelligent reader, or they bewilder him by technicalities. From both of these faults "Sex Habits" is remarkably free. One is impressed by the authors' complete mastery of their subject; they write from a wide experience and a well-considered point of view.

The book begins with a description of the physiology of reproduction, clearly and simply stated, and well illustrated by cuts which would do credit to any medical textbook. The sexual impulse in its relation to marriage and to society, its aberrations and its abnormalities, are then considered. The concluding chapters deal with general considerations—heredity, eugenics and racial hygiene, advice concerning marriage, and monogamy.

While all of it is admirable, certain sections impressed the reviewer as being especially worthy of mention. The chapter on puberty, for example, is written with such wisdom and understanding that all parents and teachers might read it with much profit.

In their discussion of the control and direction of the sex impulse, about which so much has been written and so much loose thinking has taken place, Buschke and Jacobsohn take a firm stand for self-control, with monogamy and a "single standard" as the only ideal compatible with a continuation of our civilization. In taking this position, they do not pass over lightly the inherent difficulties in such a course. They admit there is a tendency to polygamy natural to the male, and give full weight to the dilemma in which the bachelor between twenty and thirty years of age finds himself. The former they say must be controlled for the sake of the monogamous ideal; the latter problem they hope will be solved by early marriage.

Their views upon abortion will not find a sympathetic reception, especially in this country. They possess a certain logic, nevertheless. The question whether a child should be born, they maintain, belongs not to the mother, but to the State. The State "must provide institutions at which abortion can be induced by experts, not only when the continuation of pregnancy would endanger the mother's health, but also when no economic provision for the upbringing of the child is forthcoming, when the existence of heredity taint makes it almost certain that the offspring will be diseased, when the pregnancy has been the outcome of rape, of the seduction of a girl under age, and so on". The above passage seems to us the only important opinion expressed in the book to which objection may be made;

at that, certain sections of it will meet with common approval.

Medical men need not think that the book is simply a restatement of facts already well known; it is that, but it is more. It is a wise, intelligent, impartial and clear discussion of the problems which are presented to every medical man — in fact, to every human being who has reached the stage of adolescence.

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*Proceedings of the First International Congress of Mental Hygiene*, edited by Frankwood E. Williams. New York: The International Committee for Mental Hygiene, Inc., 1932. 2 vols.

The mental-hygiene movement was founded by Clifford W. Beers, who, after three years of experiences in various mental hospitals, recovered his health in 1903. The first society was formed in New Haven, Connecticut, in 1908, the same year that Beers wrote his autobiography, A Mind that Found Itself. Twenty-one years later, national societies had been formed in twenty-two countries and in 1930 the first international congress was held in Washington, D. C. The two volumes under review consist of the proceedings of this congress and contain a wealth of material on the subject of the world organization of mental hygiene, as well as many individual papers on psychiatry, institutional, educational and social. Appended are important tables giving the statistics of persons in hospitals for mental disease in thirty-two countries. There is a useful index.

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*Neurology.* By Roy R. Grinker. Springfield, Ill. and Baltimore, Md. Charles C. Thomas, 1934. 979 Pages. Price, $8.50.

This textbook of neurology, written largely for the student of medicine, is an important departure from the old standard type of book and, in spite of some well-marked deficiencies, is a useful addition to neurological literature. The author, an associate professor at the University of Chicago, has drawn his clinical material from the out-patient department and the hospital associated with the medical department of the same University. Far more than the average clinician or teacher, Dr. Grinker views neurology as a special field in the biological sciences and, therefore, his book is based upon a thorough consideration of the embryology, anatomy and physiology of the nervous system. This feature greatly adds to the bulk of his volume, which runs to nearly a thousand pages, and seems to the reviewer its weakest point. Granting his conclusions as sound regarding the value of the biological approach to clinical neurology, much of his material can easily be found elsewhere in books that a student *must* buy. Is the duplication worth while? If this material, valuable as it is, were removed, the book would not exceed five hundred pages.

The remainder, the clinical descriptions of diseases and syndromes, is far from satisfactory. The chapter on brain tumors is good, largely drawn from Cushing and Bailey; that on inflammatory diseases, excellent; on trauma, adequate; on syphilis, badly written and shows a poor conception of the modern treatment. On the whole, the section on clinical neurology is not sound and shows a marked lack of personal experience when compared with the classic textbooks of Gowers, Oppenheim and even with Osler's *Medicine*.

Each chapter in the book is provided with a short list of references, not always the most important. The illustrations are reasonably adequate and the text well printed. In general, however, one feels that much effort has been wasted on a project ill-conceived and not well executed. Would that the author had limited his book to the biological aspect of neurology and left the clinical field to the period of his riper experience!

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*Amerika und die Medizin.* By Henry E. Sigerist, M.D. Leipzig: Georg Thieme, 1933. 352 Pages.

To see ourselves as others see us is always a salutary process. The quick, perceiving foreign mind often notes both our strength and weakness in a way impossible for those of us who are part of medicine in America. Professor Sigerist, recently added to the staff of the Johns Hopkins University, has written a stimulating book on American medicine, which cannot fail to interest physicians and educators in this country. His detached viewpoint, broad and sympathetic understanding, wide knowledge and prodigious industry make his book an outstanding contribution to the literature.

The subject is viewed from many angles. About one-half of the book is historical: Indian medicine, the colonial period, the pioneers and great men of American medicine (Morgan, Rush, McDowell, Drake, Beaumont, Gross, Sims, Holmes, Mitchell, Billings and Osler). Nothing of importance is missed and no finer brief biographies of our medical worthies have appeared in print than these of Dr. Sigerist's. The group of hospitals, from the Philadelphia General to the great medical centers of New York, form another chapter, while the private clinics, the Mayos, doctors' office buildings and hospitals are not omitted. Medical schools, the curricula, numbers of students, the cost of education, medical literature, birth control, hygiene, and similar topics are discussed, often in some detail.

The illustrations consist of excellent portraits of American physicians, pictures of hospitals and medical schools, tables of important statistics, and a folding map of the United States. The indexes, unfortunately, are much abbreviated. The printing and general appearance of the book leave nothing to be desired.

In general, one feels that this book is a scholarly presentation of the subject. Written mainly for European readers, the work, nevertheless, is an important asset to American medicine. It is worthy of an English translation.